

=> d his nofile

(FILE 'HOME' ENTERED AT 09:52:22 ON 21 NOV 2006)

FILE 'HCAPLUS' ENTERED AT 09:52:35 ON 21 NOV 2006

E US2005-532447/APPS

L1 1 SEA ABB=ON PLU=ON US2005-532447/AP

E WO2003-EP12584/APPS

L2 1 SEA ABB=ON PLU=ON (WO2003-EP12584/AP OR WO2003-EP12584/PRN)

L3 1 SEA ABB=ON PLU=ON (L1 OR L2)

D SCAN

FILE 'REGISTRY' ENTERED AT 09:53:43 ON 21 NOV 2006

E TRIIODOTHYRONINE SULFATE/CN

L4 1 SEA ABB=ON PLU=ON "TRIIODOTHYRONINE SULFATE"/CN

D BROWSE

FILE 'REGISTRY' ENTERED AT 09:54:27 ON 21 NOV 2006

L5 STR 31135-55-4

L6 3 SEA FAM FUL L5

D SCAN

L7 0 SEA ABB=ON PLU=ON 31135-55-4/CRN

FILE 'HCAPLUS' ENTERED AT 09:55:10 ON 21 NOV 2006

L8 90 SEA ABB=ON PLU=ON L6

E TRIIODOTHYRONINE SULFATE/CT

L9 61 SEA ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?

L10 6 SEA ABB=ON PLU=ON L6 (L) (THU OR PKT OR DMA OR PAC OR

BAC)/RL

D KWIC

FILE 'STNGUIDE' ENTERED AT 09:57:04 ON 21 NOV 2006

FILE 'HCAPLUS' ENTERED AT 09:57:42 ON 21 NOV 2006

E HYPOTHYROIDISM/CT

E E3+ALL

L11 6796 SEA ABB=ON PLU=ON HYPOTHYROIDISM/CT

E HYPOTHYROIDISM/CT

E E4+ALL

L12 363 SEA ABB=ON PLU=ON "HYPOTHYROIDISM (L) CONGENITAL"/CT

E HYPOTHYROIDISM/CT

E E5+ALL

L13 231 SEA ABB=ON PLU=ON "HYPOTHYROIDISM (L) CRETINISM"+OLD/CT

E HYPOTHYROIDISM/CT

E E6+ALL

L14 568 SEA ABB=ON PLU=ON "HYPOTHYROIDISM (L) MYXEDEMA"+OLD/CT

E HYPOTHYROIDISM/CT

E E7+ALL

E THYROID GLAND/CT

E E3+ALL

L15 37088 SEA ABB=ON PLU=ON "THYROID GLAND"+OLD/CT

E THYROID GLAND/CT

E E9+ALL

E E2+ALL

L16 2460 SEA ABB=ON PLU=ON "AUTOIMMUNE DISEASE (L) THYROID"+OLD/CT

E E11+ALL

E THYROID GLAND/CT

E E9+ALL

E E3+ALL

L17 2433 SEA ABB=ON PLU=ON "THYROID GLAND, DISEASE (L) AUTOIMMUNE"+OLD

```

/CT
E THYROID GLAND/CT
E E10+ALL
E E2+ALL
L18      1718 SEA ABB=ON  PLU=ON  "AUTOIMMUNE DISEASE (L) AUTOIMMUNE
          THYROIDITIS"+OLD/CT
          E THYROID GLAND/CT
          E E10+ALL
          E E3+ALL
L19      1718 SEA ABB=ON  PLU=ON  "INFLAMMATION (L) AUTOIMMUNE THYROIDITIS"+O
          LD/CT
          E THYROID GLAND/CT
          E E10+ALL
          E E4+ALL
L20      1718 SEA ABB=ON  PLU=ON  "THYROID GLAND, DISEASE (L) AUTOIMMUNE
          THYROIDITIS"+OLD/CT
          E THYROID GLAND/CT
          E E11+ALL
          E E2+ALL
L21      2460 SEA ABB=ON  PLU=ON  "AUTOIMMUNE DISEASE (L) THYROID"+OLD/CT
          E THYROID GLAND/CT
          E E11+ALL
          E E3+ALL
L22      2433 SEA ABB=ON  PLU=ON  "THYROID GLAND, DISEASE (L) AUTOIMMUNE"+OLD
          /CT
          E THYROID GLAND/CT
          E E26+ALL
          E THYROIDECTOMY/CT
          E E3+ALL
          E E2+ALL
L23      1578 SEA ABB=ON  PLU=ON  "SURGERY (L) THYROIDECTOMY"+OLD/CT
          E THYROIDECTOMY/CT
          E E3+ALL
          E E3+ALL
L24      1595 SEA ABB=ON  PLU=ON  "THYROID GLAND (L) THYROIDECTOMY"+OLD/CT
L*** DEL 86497 S (HYPOTHYROID? OR THYROID?)
L*** DEL 67  S L11-L25 AND L8-L10
          D KWIC
L*** DEL 11987 S HYPOTHYROID?
L25      11987 SEA ABB=ON  PLU=ON  HYPOTHYROID?
L26      15484 SEA ABB=ON  PLU=ON  (THYROID?) (L) (DISEASE? OR DISORDER? OR
          DYSFUNCTION? OR AUTOIMMUN?)
L27      22  SEA ABB=ON  PLU=ON  (L8 OR L9 OR L10) AND (L11 OR L12 OR L13
          OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22
          OR L23 OR L24 OR L25 OR L26)
          D KWIC
          D KWIC 2
          D KWIC 3
          D KWIC 4
          D KWIC 5
          D KWIC 6
          D KWIC 7
          D KWIC 8
          D KWIC 9
          D KWIC 10
L28      24  SEA ABB=ON  PLU=ON  (L27 OR L10)
L29      24  SEA ABB=ON  PLU=ON  (L28 OR L3)
L30      23  SEA ABB=ON  PLU=ON  L29 NOT L3
L31      2145712 SEA ABB=ON  PLU=ON  PHARMAC?/SC,SX
L32      11  SEA ABB=ON  PLU=ON  L31 AND (L8 OR L9 OR L10)

```

L33 4 SEA ABB=ON PLU=ON L32 AND (L11 OR L12 OR L13 OR L14 OR L15
 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23 OR L24
 OR L25 OR L26)
 L34 31 SEA ABB=ON PLU=ON (L32 OR L33 OR L30)
 L35 30 SEA ABB=ON PLU=ON L34 NOT L3
 D SCAN TI
 E PINCHERA A/AU
 L36 271 SEA ABB=ON PLU=ON ("PINCHERA A"/AU OR "PINCHERA ALDO"/AU)
 E SANTINI F/AU
 L37 106 SEA ABB=ON PLU=ON ("SANTINI F"/AU OR "SANTINI F G"/AU OR
 "SANTINI F J"/AU OR "SANTINI FERRUCCIO"/AU OR "SANTINI
 FERRUCIO"/AU)
 L38 23 SEA ABB=ON PLU=ON L36 AND L37
 L39 30 SEA ABB=ON PLU=ON L35 NOT L38

FILE 'MEDLINE' ENTERED AT 10:12:24 ON 21 NOV 2006

L40 31 SEA ABB=ON PLU=ON L6
 E TRIIODOTHYRONINE SULFATE/CT
 L41 35 SEA ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
 E HYPOTHYROIDISM/CT
 E E3+ALL
 L42 23512 SEA ABB=ON PLU=ON HYPOTHYROIDISM+NT/CT
 E THYROID GLAND/CT
 E E3+ALL
 L43 40322 SEA ABB=ON PLU=ON "THYROID GLAND"/CT
 E THYROID GLAND/CT
 L44 37881 SEA ABB=ON PLU=ON (THYROID?) (L) (DISEASE? OR DISORDER? OR
 DYSFUNCTION? OR AUTOIMMUN?)
 L45 27381 SEA ABB=ON PLU=ON HYPOTHYROID?
 E THYROIDECTOMY/CT
 E E3+ALL
 L46 12721 SEA ABB=ON PLU=ON THYROIDECTOMY/CT
 L47 15323 SEA ABB=ON PLU=ON THYROIDECTOMY?
 L48 11 SEA ABB=ON PLU=ON (L40 OR L41) AND (L42 OR L43 OR L44 OR L45
 OR L46 OR L47)
 D KWIC
 D KWIC 2
 D KWIC 3
 D KWIC 4
 D KWIC 5
 D KWIC 6
 D KWIC 7
 E DRUG COMPOSITIONS/CT
 E PHARMACUETICALS/CT
 L49 0 SEA ABB=ON PLU=ON (L40 OR L41) (L) TU/CT
 L50 1251950 SEA ABB=ON PLU=ON TU/CT
 L51 2 SEA ABB=ON PLU=ON L50 AND (L40 OR L41)
 D KWIC
 L52 17 SEA ABB=ON PLU=ON (PK OR PD)/CT AND (L40 OR L41)
 D KWIC
 D KWIC 2
 L53 4 SEA ABB=ON PLU=ON L52 AND (L42 OR L43 OR L44 OR L45 OR L46
 OR L47)
 L54 12 SEA ABB=ON PLU=ON (L53 OR L51 OR L48)
 L55 13 SEA ABB=ON PLU=ON L52 NOT L54
 D KWIC
 D KWIC 2
 D KWIC 3
 D KWIC 4

L56 0 SEA ABB=ON PLU=ON L55 AND (PY<2003 AND AY<2003 OR PRY<2003)

FILE 'EMBASE' ENTERED AT 10:22:10 ON 21 NOV 2006

L57 0 SEA ABB=ON PLU=ON L6
E TRIIODOTHYRONINE SULFATE/CT
L58 23 SEA ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
L59 28070 SEA ABB=ON PLU=ON (THYROID?) (L) (DISEASE? OR DISORDER? OR
DYSFUNCTION? OR AUTOIMMUN?)
L60 10321 SEA ABB=ON PLU=ON THYROIDECTOMY?
L61 24179 SEA ABB=ON PLU=ON HYPOTHYROID?
L62 7 SEA ABB=ON PLU=ON L58 AND (L59 OR L60 OR L61)
D KWIC
L63 0 SEA ABB=ON PLU=ON L62 AND (PY<2003 AND AY<2003 OR PRY<2003)
D BIB 1-7
D BIB 1-7 L62
E HYPOTHYROID/CT
E THYROID GLAND/CT
E E3+ALL
L64 12996 SEA ABB=ON PLU=ON "THYROID GLAND"/CT
L65 17379 SEA ABB=ON PLU=ON "THYROID GLAND"+NT/CT
E THYROIDECTOMY/CT
E E3+ALL
L66 8213 SEA ABB=ON PLU=ON THYROIDECTOMY/CT
E THYROIDECTOMY/CT
E E4+ALL
E E2+ALL
L67 905 SEA ABB=ON PLU=ON "SUBTOTAL THYROIDECTOMY"/CT
L68 2 SEA ABB=ON PLU=ON L58 AND (L66 OR L67)
L69 7 SEA ABB=ON PLU=ON (L68 OR L62)

FILE 'MEDLINE' ENTERED AT 10:25:37 ON 21 NOV 2006

D BIB L55 1-13
D SCAN L55
D TRIAL L55
D TRIAL L55 2
D TRIAL L55 3
D TRIAL L55 4
D TRIAL L55 5
D TRIAL L55 6
D TRIAL L55 7
D TRIAL L55 8
D TRIAL L55 9
D TRIAL L55 10

FILE 'BIOSIS' ENTERED AT 10:29:08 ON 21 NOV 2006

L70 18 SEA ABB=ON PLU=ON L6
L71 35 SEA ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
L72 35193 SEA ABB=ON PLU=ON (THYROID?) (L) (DISEASE? OR DISORDER? OR
DYSFUNCTION? OR AUTOIMMUN?)
L73 16164 SEA ABB=ON PLU=ON HYPOTHYROID?
L74 5577 SEA ABB=ON PLU=ON THYROIDECTOMY?
L75 9 SEA ABB=ON PLU=ON (L70 OR L71) AND (L72 OR L73 OR L74)
D KWIC
D KWIC 2

FILE 'WPIX' ENTERED AT 10:31:21 ON 21 NOV 2006

L76 0 SEA SSS SAM L5
E TRIIODOTHYRONINE SULFATE/CN
L77 1 SEA ABB=ON PLU=ON "TRIIODOTHYRONINE SULFATE"/CN
D TOT SDCN DCSE

L78 1 SEA ABB=ON PLU=ON RAEEDH/DCN
 L79 0 SEA ABB=ON PLU=ON 908404-1-0-0/DCRE
 L80 2 SEA ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?/BIX
 L81 2 SEA ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?/BIX,ABEX
 L82 3 SEA ABB=ON PLU=ON (L76 OR L77 OR L78 OR L79 OR L80 OR L81)
 L83 5624 SEA ABB=ON PLU=ON ((THYROID?) (L) (DISEASE? OR DISORDER? OR
 DYSFUNCTION? OR AUTOIMMUN?))/BIX,ABEX
 L84 648 SEA ABB=ON PLU=ON HYPOTHYROID?/BIX,ABEX
 L85 18 SEA ABB=ON PLU=ON THYROIDECTOMY?/BIX,ABEX
 L86 2 SEA ABB=ON PLU=ON L82 AND (L83 OR L84 OR L85)

FILE 'STNGUIDE' ENTERED AT 10:34:16 ON 21 NOV 2006

FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS, WPIX' ENTERED AT 10:34:30 ON 21 NOV 2006

L87 41 DUP REM L35 L54 L69 L75 L86 (19 DUPLICATES REMOVED)
 ANSWERS '1-30' FROM FILE HCAPLUS
 ANSWERS '31-37' FROM FILE MEDLINE
 ANSWER '38' FROM FILE EMBASE
 ANSWERS '39-40' FROM FILE BIOSIS
 ANSWER '41' FROM FILE WPIX

=> file hcaplus

FILE 'HCAPLUS' ENTERED AT 10:34:51 ON 21 NOV 2006

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

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FILE COVERS 1907 - 21 Nov 2006 VOL 145 ISS 22

FILE LAST UPDATED: 20 Nov 2006 (20061120/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> file medline

FILE 'MEDLINE' ENTERED AT 10:34:54 ON 21 NOV 2006

FILE LAST UPDATED: 15 Nov 2006 (20061115/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).

See also:

<http://www.nlm.nih.gov/mesh/>

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> file embase

FILE 'EMBASE' ENTERED AT 10:34:56 ON 21 NOV 2006

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FILE COVERS 1974 TO 20 Nov 2006 (20061120/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> file biosis

FILE 'BIOSIS' ENTERED AT 10:34:57 ON 21 NOV 2006

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FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 15 November 2006 (20061115/ED)

=> file wpix

FILE 'WPIX' ENTERED AT 10:35:00 ON 21 NOV 2006

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FILE LAST UPDATED: 20 NOV 2006 <20061120/UP>

MOST RECENT THOMSON SCIENTIFIC UPDATE: 200674 <200674/DW>

DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> YOU ARE IN THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX <<<

>>> FOR DETAILS ON THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX

PLEASE VISIT:

http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<

FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,

PLEASE VISIT:

http://www.stn-international.de/training_center/patents/stn_guide.pdf

FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE

<http://scientific.thomson.com/support/patents/coverage/latestupdates/>

PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE

http://www.stn-international.de/stndatabases/details/ipc_reform.html and

<http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf>

>>> FOR DETAILS ON THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX
PLEASE SEE
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<

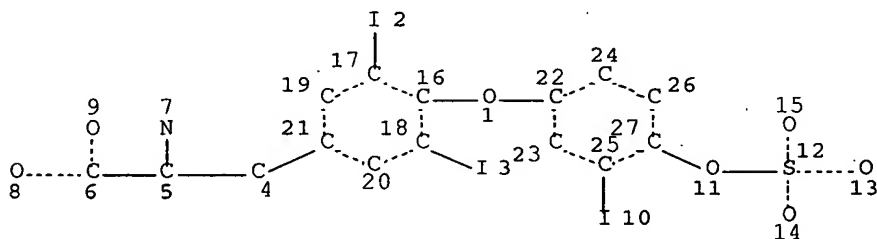
>>> YOU ARE IN THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX <<<
'BIX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE

=> d que l38

L36 271 SEA FILE=HCAPLUS ABB=ON PLU=ON ("PINCHERA A"/AU OR "PINCHERA
ALDO"/AU)
L37 106 SEA FILE=HCAPLUS ABB=ON PLU=ON ("SANTINI F"/AU OR "SANTINI F
G"/AU OR "SANTINI F J"/AU OR "SANTINI FERRUCCIO"/AU OR
"SANTINI FERRUCCIO"/AU)
L38 23 SEA FILE=HCAPLUS ABB=ON PLU=ON L36 AND L37

=> d que l35

L1 1 SEA FILE=HCAPLUS ABB=ON PLU=ON US2005-532447/AP
L2 1 SEA FILE=HCAPLUS ABB=ON PLU=ON (WO2003-EP12584/AP OR
WO2003-EP12584/PRN)
L3 1 SEA FILE=HCAPLUS ABB=ON PLU=ON (L1 OR L2)
L5 STR



NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 27

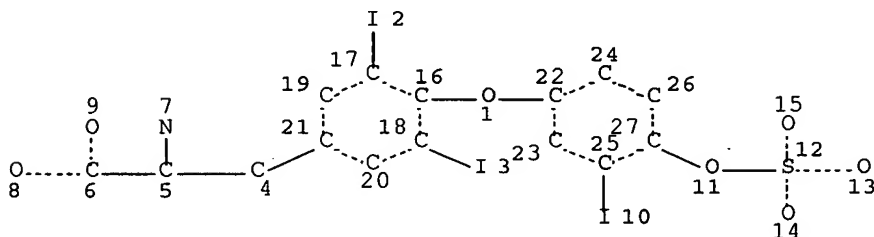
STEREO ATTRIBUTES: NONE

L6 3 SEA FILE=REGISTRY FAM FUL L5
L8 90 SEA FILE=HCAPLUS ABB=ON PLU=ON L6
L9 61 SEA FILE=HCAPLUS ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
L10 6 SEA FILE=HCAPLUS ABB=ON PLU=ON L6 (L) (THU OR PKT OR DMA OR
PAC OR BAC)/RL
L11 6796 SEA FILE=HCAPLUS ABB=ON PLU=ON HYPOTHYROIDISM/CT
L12 363 SEA FILE=HCAPLUS ABB=ON PLU=ON "HYPOTHYROIDISM (L) CONGENITAL
"/CT
L13 231 SEA FILE=HCAPLUS ABB=ON PLU=ON "HYPOTHYROIDISM (L) CRETINISM"
+OLD/CT
L14 568 SEA FILE=HCAPLUS ABB=ON PLU=ON "HYPOTHYROIDISM (L) MYXEDEMA"+
OLD/CT

L15	37088	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"THYROID GLAND"+OLD/CT
L16	2460	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"AUTOIMMUNE DISEASE (L) THYROID"+OLD/CT
L17	2433	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"THYROID GLAND, DISEASE (L) AUTOIMMUNE"+OLD/CT
L18	1718	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"AUTOIMMUNE DISEASE (L) AUTOIMMUNE THYROIDITIS"+OLD/CT
L19	1718	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"INFLAMMATION (L) AUTOIMMUNE THYROIDITIS"+OLD/CT
L20	1718	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"THYROID GLAND, DISEASE (L) AUTOIMMUNE THYROIDITIS"+OLD/CT
L21	2460	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"AUTOIMMUNE DISEASE (L) THYROID"+OLD/CT
L22	2433	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"THYROID GLAND, DISEASE (L) AUTOIMMUNE"+OLD/CT
L23	1578	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"SURGERY (L) THYROIDECTOMY"+OL D/CT
L24	1595	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	"THYROID GLAND (L) THYROIDECTO MY"+OLD/CT
L25	11987	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	HYPOTHYROID?
L26	15484	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	(THYROID?) (L) (DISEASE? OR DISORDER? OR DYSFUNCTION? OR AUTOIMMUN?)
L27	22	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	(L8 OR L9 OR L10) AND (L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26)
L28	24	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	(L27 OR L10)
L29	24	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	(L28 OR L3)
L30	23	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	L29 NOT L3
L31	2145712	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	PHARMAC?/SC,SX
L32	11	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	L31 AND (L8 OR L9 OR L10)
L33	4	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	L32 AND (L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26)
L34	31	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	(L32 OR L33 OR L30)
L35	30	SEA FILE=HCAPLUS	ABB=ON	PLU=ON	L34 NOT L3

=> d que 154

L5 STR



NODE ATTRIBUTES:

DEFAULT MLEVEL IS ATOM

DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:

RING(S) ARE ISOLATED OR EMBEDDED

NUMBER OF NODES IS 27

STEREO ATTRIBUTES: NONE

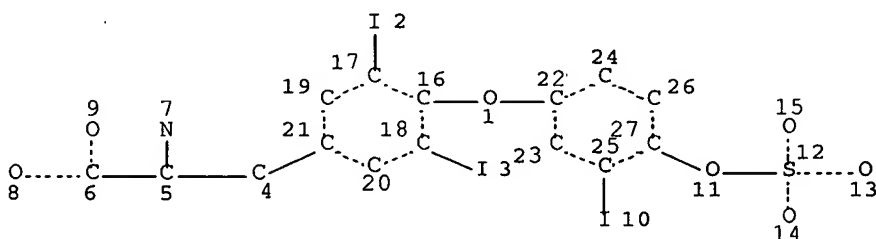
L6 3 SEA FILE=REGISTRY FAM FUL L5
L40 31 SEA FILE=MEDLINE ABB=ON PLU=ON L6
L41 35 SEA FILE=MEDLINE ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
L42 23512 SEA FILE=MEDLINE ABB=ON PLU=ON HYPOTHYROIDISM+NT/CT
L43 40322 SEA FILE=MEDLINE ABB=ON PLU=ON "THYROID GLAND"/CT
L44 37881 SEA FILE=MEDLINE ABB=ON PLU=ON (THYROID?) (L) (DISEASE? OR
DISORDER? OR DYSFUNCTION? OR AUTOIMMUN?)
L45 27381 SEA FILE=MEDLINE ABB=ON PLU=ON HYPOTHYROID?
L46 12721 SEA FILE=MEDLINE ABB=ON PLU=ON THYROIDECTOMY/CT
L47 15323 SEA FILE=MEDLINE ABB=ON PLU=ON THYROIDECTOMY?
L48 11 SEA FILE=MEDLINE ABB=ON PLU=ON (L40 OR L41) AND (L42 OR L43
OR L44 OR L45 OR L46 OR L47)
L50 1251950 SEA FILE=MEDLINE ABB=ON PLU=ON TU/CT
L51 2 SEA FILE=MEDLINE ABB=ON PLU=ON L50 AND (L40 OR L41)
L52 17 SEA FILE=MEDLINE ABB=ON PLU=ON (PK OR PD)/CT AND (L40 OR
L41)
L53 4 SEA FILE=MEDLINE ABB=ON PLU=ON L52 AND (L42 OR L43 OR L44 OR
L45 OR L46 OR L47)
L54 12 SEA FILE=MEDLINE ABB=ON PLU=ON (L53 OR L51 OR L48)

=> d que 169

L58 23 SEA FILE=EMBASE ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
L59 28070 SEA FILE=EMBASE ABB=ON PLU=ON (THYROID?) (L) (DISEASE? OR
DISORDER? OR DYSFUNCTION? OR AUTOIMMUN?)
L60 10321 SEA FILE=EMBASE ABB=ON PLU=ON THYROIDECTOMY?
L61 24179 SEA FILE=EMBASE ABB=ON PLU=ON HYPOTHYROID?
L62 7 SEA FILE=EMBASE ABB=ON PLU=ON L58 AND (L59 OR L60 OR L61)
L66 8213 SEA FILE=EMBASE ABB=ON PLU=ON THYROIDECTOMY/CT
L67 905 SEA FILE=EMBASE ABB=ON PLU=ON "SUBTOTAL THYROIDECTOMY"/CT
L68 2 SEA FILE=EMBASE ABB=ON PLU=ON L58 AND (L66 OR L67)
L69 7 SEA FILE=EMBASE ABB=ON PLU=ON (L68 OR L62)

=> d que 175

L5 STR



NODE ATTRIBUTES:

DEFAULT MLEVEL IS ATOM

DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:

RING(S) ARE ISOLATED OR EMBEDDED

NUMBER OF NODES IS 27

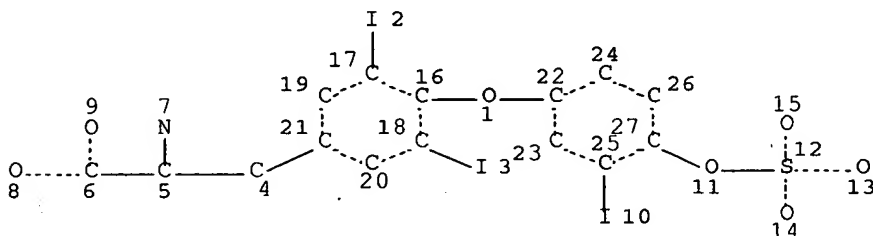
STEREO ATTRIBUTES: NONE

L6 3 SEA FILE=REGISTRY FAM FUL L5

L70 18 SEA FILE=BIOSIS ABB=ON PLU=ON L6
 L71 35 SEA FILE=BIOSIS ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?
 L72 35193 SEA FILE=BIOSIS ABB=ON PLU=ON (THYROID?) (L) (DISEASE? OR
 DISORDER? OR DYSFUNCTION? OR AUTOIMMUN?)
 L73 16164 SEA FILE=BIOSIS ABB=ON PLU=ON HYPOTHYROID?
 L74 5577 SEA FILE=BIOSIS ABB=ON PLU=ON THYROIDECTOMY?
 L75 9 SEA FILE=BIOSIS ABB=ON PLU=ON (L70 OR L71) AND (L72 OR L73
 OR L74)

=> d que 186

L5 STR



NODE ATTRIBUTES:

DEFAULT MLEVEL IS ATOM

DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:

RING(S) ARE ISOLATED OR EMBEDDED

NUMBER OF NODES IS 27

STEREO ATTRIBUTES: NONE

L76 0 SEA FILE=WPIX SSS SAM L5
 L77 1 SEA FILE=WPIX ABB=ON PLU=ON "TRIIODOTHYRONINE SULFATE"/CN
 L78 1 SEA FILE=WPIX ABB=ON PLU=ON RAEEDH/DCN
 L79 0 SEA FILE=WPIX ABB=ON PLU=ON 908404-1-0-0/DCRE
 L80 2 SEA FILE=WPIX ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?/BIX
 L81 2 SEA FILE=WPIX ABB=ON PLU=ON TRIIODOTHYRONINE SULFATE?/BIX,ABE
 X
 L82 3 SEA FILE=WPIX ABB=ON PLU=ON (L76 OR L77 OR L78 OR L79 OR L80
 OR L81)
 L83 5624 SEA FILE=WPIX ABB=ON PLU=ON ((THYROID?) (L) (DISEASE? OR
 DISORDER? OR DYSFUNCTION? OR AUTOIMMUN?))/BIX,ABEX
 L84 648 SEA FILE=WPIX ABB=ON PLU=ON HYPOTHYROID?/BIX,ABEX
 L85 18 SEA FILE=WPIX ABB=ON PLU=ON THYROIDECTOMY?/BIX,ABEX
 L86 2 SEA FILE=WPIX ABB=ON PLU=ON L82 AND (L83 OR L84 OR L85)

=> dup rem 138,135,154,169,175,186

FILE 'HCAPLUS' ENTERED AT 10:35:39 ON 21 NOV 2006

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PROCESSING COMPLETED FOR L38
PROCESSING COMPLETED FOR L35
PROCESSING COMPLETED FOR L54
PROCESSING COMPLETED FOR L69
PROCESSING COMPLETED FOR L75
PROCESSING COMPLETED FOR L86

L88 63 DUP REM L38 L35 L54 L69 L75 L86 (20 DUPLICATES REMOVED)
 ANSWERS '1-53' FROM FILE HCAPLUS
 ANSWERS '54-60' FROM FILE MEDLINE
 ANSWER '61' FROM FILE EMBASE
 ANSWERS '62-63' FROM FILE BIOSIS

=> d ibib abs hitind retable l88 1-53;d iall l88 54-63

L88 ANSWER 1 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:430742 HCAPLUS Full-text
DOCUMENT NUMBER: 140:412353
TITLE: 3,5,3'-tri-iodothyronine sulfate as thyromimetic agent
 and pharmaceutical formulations thereof
INVENTOR(S): *Pinchera, Aldo; Santini, Ferruccio*
PATENT ASSIGNEE(S): Bracco S.p.A., Italy
SOURCE: PCT Int. Appl., 17 pp.
 CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004043452	A1	20040527	WO 2003-EP12584	20031111
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
AU 2003292006	A1	20040603	AU 2003-292006	20031111
EP 1560575	A1	20050810	EP 2003-767529	20031111
EP 1560575	B1	20060927		
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			
CN 1711079	A	20051221	CN 2003-80103057	20031111
JP 2006508956	T2	20060316	JP 2004-550983	20031111
AT 340570	E	20061015	AT 2003-767529	20031111
US 2005272816	A1	20051208	US 2005-532447	20050422
PRIORITY APPLN. INFO.:			IT 2002-MI2394	A 20021113
			WO 2003-EP12584	W 20031111
AB	The invention regards the use of tri-iodothyronine sulfate, commonly named T3S, as a medicament having thyromimetic activity for the treatment of			

pathologies due to organic deficiency of triiodothyronine (T3), as such or in association with thyroxine (T4), and pharmaceutical formulations thereof.

IC ICM A61K031-198

ICS A61P005-14

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 2

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Chopra, I	1996	6	229	THYROID 1996 UNITED	MEDLINE
Santini, F	1993	133	105	ENDOCRINOLOGY	HCAPLUS

L88 ANSWER 2 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2002:90345 HCAPLUS Full-text

DOCUMENT NUMBER: 136:145560

TITLE: Method for diagnosing thyroid conditions and for monitoring thyroxine therapy

INVENTOR(S): Salhanick, Hilton A.; Hourihan, Joachim

PATENT ASSIGNEE(S): Biodiagnostics, Inc., USA

SOURCE: PCT Int. Appl., 114 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002008759	A1	20020131	WO 2001-US23593	20010726
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
EP 1307741	A1	20030507	EP 2001-955988	20010726
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR			

PRIORITY APPLN. INFO.: US 2000-220894P P 20000726

WO 2001-US23593 W 20010726

AB This invention provides a method of diagnosing a thyroid condition in a subject which comprises: determining the concentration of TSH in a urine sample by a method which is not a RIA; and comparing the concentration of TSH with a urinary concentration of TSH in a normal subject; wherein: a concentration of TSH which is higher than the urinary concentration of TSH in the normal subject diagnoses *hypothyroidism* in the subject; and a concentration of TSH which is lower than the urinary concentration of TSH in the normal subject diagnoses hyperthyroidism in the subject. This invention also proves a method of monitoring thyroxine therapy.

IC ICM G01N033-53

ICS G01N033-566

CC 2-1 (Mammalian Hormones)

Section cross-reference(s): 14

ST **thyroid gland disease** diagnosis thyroxine treatment
monitoring assay kit

IT Colorimetric indicators

Diagnosis
 Enzyme immunoassay
 Fluorescent indicators
 Human
 Hyperthyroidism
Hypothyroidism
 Immobilization, molecular or cellular
 Polarized fluorescence
Thyroid gland, disease

Urine analysis

(method for diagnosing *thyroid* conditions and for monitoring
 thyroxine therapy by measurement of *thyroid* gland
 hormones/thyroxine levels utilizing non-RIA assay systems and/or kits)

IT 6893-02-3, Triiodothyronine 9002-71-5, TSH 21462-56-6, Thyroxine
 glucuronide 29919-72-0, Triiodothyronine glucuronide 31135-55-4
 , Triiodothyronine sulfate 77074-49-8, Thyroxine
 sulfate

RL: BSU (Biological study, unclassified); *THU (Therapeutic use)*;

BIOL (Biological study); USES (Uses)

(method for diagnosing thyroid conditions and for monitoring thyroxine
 therapy by measurement of thyroid gland hormones/thyroxine levels
 utilizing non-RIA assay systems and/or kits)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Aghini-Lombardi	1999	84		Journal Of Clinical	HCAPLUS
Black	1981	2	443	Lancet	HCAPLUS
Knudsen	1999	51	361	Clinical Endocrinolo	MEDLINE
Premachandra	1982	78	63	American Journal Of	MEDLINE
Wu	1997			US 5670380 A	HCAPLUS

L88 ANSWER 3 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3

ACCESSION NUMBER: 1998:377674 HCAPLUS Full-text

DOCUMENT NUMBER: 129:145038

TITLE: Urinary compound W in pregnant women is a potential
 marker for fetal thyroid function

AUTHOR(S): Wu, Sing-Yung; Fisher, Delbert A.; Huang, Wen-Sheng;
 Beck-Peccoz, Paolo; Emerson, Charles H.; Kuo, Shi-Wen;
 Chen, Wei-Lian

CORPORATE SOURCE: Veterans Administration Medical Center, the Perinatal
 Laboratory, Nuclear Medicine and Medical Services, Los
 Angeles, CA, USA

SOURCE: American Journal of Obstetrics and Gynecology (1998),
 178(5), 886-891

CODEN: AJOGAH; ISSN: 0002-9378

PUBLISHER: Mosby, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Previously the authors reported 3,3'-diiodothyronine sulfate-like material
 (compound W) in maternal serum, and studies suggest that compound W is derived
 from thyroid hormones of fetal origin. In this study the authors
 characterized gestational changes of urinary compound W concns. to correlate
 with changes in serum concns. Urinary samples were collected from 94 women at
 various gestational ages ranging from 3 to 40 wk. Urinary compound W was
 first identified biochem. The concns. of compound W (adjusted for creatinine
 levels) were assessed by a 3,3'-diiodothyronine sulfate radioimmunoassay in
 ethanol exts. of urine samples. Compound W increased to 88 pmol (of 3,3'-
 diiodothyronine sulfate equivalent)/mmol creatinine in urinary samples
 obtained from 26 women in the first trimester of pregnancy compared with 40

pmol/mmol creatinine in 10 nonpregnant women. Excretion of compound W increased further during the second and third trimesters: 171 and 434 resp. In contrast, urinary 3,3',5-triiodothyronine sulfate concns. measured by RIA were similar during pregnancy to values in nonpregnant women. Urinary compound W concns. increase with the progression of normal pregnancy and correlate with the increase in serum levels. Random spot urine compound W concns., adjusted for creatinine levels, may be used in place of serum levels in conditions in which obtaining serum samples may be tech. difficult, especially during population screening.

CC 2-7 (Mammalian Hormones)

IT Blood serum

Pregnancy

Thyroid gland

Urine

(urinary compound W in pregnant women as potential marker for fetal thyroid function)

IT 31135-55-4, Triiodothyronine sulfate

64192-57-0, 3,3'-Diiodothyronine sulfate

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(urinary compound W in pregnant women as potential marker for fetal thyroid function)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abuhamad, A	1995	6	368	Ultrasound Obstet Gy	MEDLINE
Beck-Peccoz, P	1995	5	S203	Thyroid	
Chopra, I	1992	75	189	J Clin Endocrinol Me	MEDLINE
Daffos, F	1989	40	319	Annu Rev Med	MEDLINE
Eelkman-Rooda, S	1988	9	125	J Immunoassay	MEDLINE
Glorieux, J	1988	24	6	Pediatr Res	MEDLINE
Hetzel, B	1994		1	The damaged brain of	
Kirk, R	1982		112	Experimental design	
Kirschenbaum, M	1981		29	Practical nephrology	
Mol, J	1985	117	1	Endocrinology	HCAPLUS
Moses, A	1996		628	The thyroid 7th ed	
Porterfield, S	1993	14	94	Endocrinol Rev	HCAPLUS
Vanmiddlesworth, L	1995	5	S106	Thyroid	
Vestergaard, P	1958	51	211	J Lab Clin Med	HCAPLUS
Wu, S	1993	265	E115	Am J Physiol, Endocr	
Wu, S	1995	268	E33	Am J Physiol, Endocr	
Wu, S	1992	131	1751	Endocrinology	HCAPLUS
Wu, S	1976	43	682	J Clin Endocrinol Me	HCAPLUS
Wu, S	1993	76	1625	J Clin Endocrinol Me	HCAPLUS
Wu, S	1994	78	1505	J Clin Endocrinol Me	HCAPLUS
Wu, S	1992	2	101	Thyroid	MEDLINE
Zar, J	1984		306	Biostatistical analy	

L88 ANSWER 4 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5

ACCESSION NUMBER: 1996:75140 HCAPLUS Full-text

TITLE: Study of serum 3,5,3'-triiodothyronine sulfate concentration in patients with systemic non-thyroidal illness

AUTHOR(S): Santini, Ferruccio; Chiovato, Luca; Bartalena, Luigi; Lapi, Paola; Palla, Roberto; Panichi, Vincenzo; Velluzzi, Fernanda; Grasso, Lucia; Chopra, Inder J.; et al.

CORPORATE SOURCE: Inst. Endocrinology, Univ. Pisa, Pisa, Italy

SOURCE: European Journal of Endocrinology (1996), 134(1), 45-9

CODEN: EJOEEP; ISSN: 0804-4643
PUBLISHER: Scandinavian University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Sulfation is an important pathway of triiodothyronine (T3) metabolism. Increased serum T3 sulfate (T3S) values have been observed during fetal life and in pathol. conditions such as hyperthyroidism and selenium deficiency. Similar variations have also been reported in a small number of patients with systemic non-*thyroidal* illness, but the underlying mechanisms have not been elucidated. In this study, serum T3S concns. have been measured by a specific RIA in 28 patients with end-stage neoplastic *disease* (ESND) and in 44 patients with chronic renal failure (CRF); 41 normal subjects served as controls. Both ESND and CRF patients had lower serum total T4 (TT4) and total T3 (TT3) than normal controls, while serum reverse T3 (rT3) was increased significantly in ESND (0.7 ± 0.5 nmol/l; $p < 0.001$ vs. controls) but not in CRF (0.3 ± 0.1 nmol/l). The TT3/rT3 ratio, an index of type I iodothyronine monodeiodinase (type I MD) activity, was reduced significantly in both groups of patients. Serum T4-binding globulin (TBG) was decreased in CRF but not in ESND patients. Serum T3S was significantly higher both in ESND (71 ± 32 pmol/l) and CRF (100 ± 24 pmol/l) than in controls (50 ± 16 pmol/l, $p < 0.001$). Serum T3S values showed a pos. correlation with rT3 values and a neg. correlation with both TT3 and FT3 values in ESND, but not in CRF. In the latter group a pos. correlation was observed between T3S and TBG values. The T3S/FT3 ratio was higher both in CRF (18 ± 5) and in ESND (23 ± 18) as compared to controls (10 ± 4). Serum inorg. sulfate was increased and correlated pos. with T3S values in CRF patients. In conclusion, the results of this study in a large series of patients confirm that patients with systemic non-*thyroidal* illness have increased serum T3S levels. The mechanisms responsible for these changes appear to be different in ESND and CRF patients. In ESND the increase in serum T3S levels is mainly related to reduced degradation of the hormone by type I MD, whereas in CRF it might be driven by the enhanced sulfate ion concentration, and could be partially dependent on the impaired renal excretion of T3S. Because T3S can be reconverted to T3, it is possible that increased T3S concns. contribute to maintenance of the euthyroid state in systemic non- *thyroidal disease*.

L88 ANSWER 5 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 1995:411911 HCAPLUS Full-text
DOCUMENT NUMBER: 122:178966
TITLE: Identification of 3,3'-T2S as a fetal thyroid hormone derivative in maternal urine in sheep
AUTHOR(S): Wu, Sing-Yung; Polk, Daniel; Fisher, Delbert A.; Huang, Wen-Sheng; Reviczky, Anita L.; Chen, Wei-Lian
CORPORATE SOURCE: Nuclear Medicine and Medical Services, Department of Veterans Affairs Medical Center, Long Beach, CA, 90822, USA
SOURCE: American Journal of Physiology (1995), 268(1, Pt. 1), E33-E39
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors measured 3,3'-diiodothyronine sulfate (T2S) in serum and urine obtained from euthyroid fetal (94-145 days of gestation, term = 150 days), newborn, and adult sheep and in serum and urine samples from ovine fetuses 13 days after total thyroidectomy conducted between 110 and 113 gestation days. Sham-operated twin fetuses served as controls. Mean serum T2S concns. increased progressively from 94 days (74 ng/dL) to 130 days (420 ng/dL), decreasing thereafter to 145 days (197 ng/dL). T2S concns. in fetal urine

peaked at 110 days (117 ng/dL). In *hypothyroid* fetuses, mean serum and urine T2S were 60 and 53% of control values. To assess the possibility that the T2S in maternal serum/urine is derived from fetal serum 3,5,3'-triiodothyronine (T3), the authors measured T3, T3 sulfate (T3S), and T2S in fetal serum and in maternal serum and urine after bolus infusion of T3 to the fetus. Addnl., T3, T3S, and T2S concns. were measured in maternal serum and urine after T3 infusion to the maternal ewes. Fetal T3 infusion rapidly increased fetal serum T3S and T2S. Maternal serum and urine T3S and T2S concns. increased, whereas T3 concns. remained unchanged. Maternal T3 infusion increased in serum and urine T3S and T2S levels, but the levels, relative to T3, were less than values measured after fetal T3 infusion. The authors conclude that T2S is a normal thyroid hormone metabolite in the ovine fetus and suggest that a major pathway of fetal T2S production is T3 to T3S to T2S. Both T3S and T2S appear to readily traverse the placenta to appear in maternal serum and urine.

CC 2-7 (Mammalian Hormones)

IT 6893-02-3, Triiodothyronine 31135-55-4, *Triiodothyronine sulfate* 64192-57-0, 3,3'-Diiodothyronine sulfate

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(diiodothyronine sulfate as a fetal thyroid hormone derivative in maternal urine in sheep)

L88 ANSWER 6 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 7

ACCESSION NUMBER: 1993:531726 HCAPLUS Full-text

DOCUMENT NUMBER: 119:131726

TITLE: The development of a radioimmunoassay for reverse *triiodothyronine sulfate* in human serum and amniotic fluid

AUTHOR(S): Wu, Sing Yung; Huang, Wen Sheng; Polk, Daniel; Chen, Wei Lian; Reviczky, Anita; Williams, John, III; Chopra, Inder J.; Fisher, Delbert A.

CORPORATE SOURCE: Nucl. Med. Med. Serv., Veterans Adm. Med. Cent., Long Beach, CA, 90822, USA

SOURCE: Journal of Clinical Endocrinology and Metabolism (1993), 76(6), 1625-30
CODEN: JCEMAZ; ISSN: 0021-972X

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Sulfated iodothyronines including T4-sulfate (T4S) and T3-sulfate (T3S) have been identified in human serum and amniotic fluid. Little is known, however, about the existence of sulfate conjugation of reverse T3 (rT3S) in man. In this report, the authors employed a novel, sensitive, and specific rT3S RIA to address this question. The rabbit antiserum to rT3S was highly specific; T4, T3, rT3, and 3,3'-T2 showed <0.002% cross-reaction with the antiserum. Only T4S and T3S cross-reacted significantly (0.3% and 0.01%, resp.); other analogs cross-reacted <0.0001%. The detection threshold of the RIA was 14 pmol/L (1.0 ng/dL). The mean serum rT3S concentration (pmol/L) was 40 in euthyroid subjects. Values were similar in *hypothyroid* patients (38) and pregnant women (52) but significantly elevated to 176 in hyperthyroid patient, 74 in patients with nonthyroid illnesses, and 684 in cord sera of newborns. Serum rT3S increased significantly in hyperthyroid patients 1 day after administration of 1 g sodium ipodate orally. Reverse T3S was detected consistently in amniotic fluid at 14 to 22 wk of gestation and showed a marked rise 1-3 wk after intraamniotic administration of 500-1000 µg T4. The various data suggest that: (1) rT3S is a normal component of human serum and amniotic fluid; (2) it is derived from metabolism of T4 or rT3; (3) circulating rT3S increases in hyperthyroidism and in circumstances where type I 5'-monodeiodinating activity is low, e.g. nonthyroid illnesses, fetal life, and after administration of ipodate.

CC 2-1 (Mammalian Hormones)
 ST reverse *triiodothyronine sulfate* detn RIA; blood serum
 rT3 sulfate detn; amniotic fluid reverse *triiodothyronine sulfate*
 IT Blood analysis
 (reverse *triiodothyronine sulfate* determination in, in
 human by RIA)
 IT Hyperthyroidism
 (reverse *triiodothyronine sulfate* of amniotic fluid
 and blood serum of humans in)
 IT Newborn
 (reverse *triiodothyronine sulfate* of blood serum of
 human)
 IT Pregnancy
 (reverse *triiodothyronine sulfate* of blood serum of
 human in)
 IT Blood serum
 (reverse *triiodothyronine sulfate* of, of human)
 IT Amniotic fluid
 (reverse *triiodothyronine sulfate* of, of human, RIA
 for determination of)
 IT Embryo
 (fetus, reverse *triiodothyronine sulfate* of blood
 serum of human)
 IT 70712-46-8
 RL: ANST (Analytical study)
 (deficiency of, reverse *triiodothyronine sulfate* of
 blood serum of human in)
 IT 77074-49-8, Thyroxine sulfate
 RL: ANST (Analytical study)
 (of amniotic fluid and blood serum of human, reverse
triiodothyronine sulfate in relation to)
 IT 5587-89-3
 RL: ANST (Analytical study)
 (reverse *triiodothyronine sulfate* of blood serum of
 human in hyperthyroidism response to)

L88 ANSWER 7 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 9

ACCESSION NUMBER: 1993:617916 HCAPLUS Full-text

DOCUMENT NUMBER: 119:217916

TITLE: Sulfate conjugates of iodothyronines in developing
 sheep: Effect of fetal *hypothyroidism*

AUTHOR(S): Wu, Sing Yung; Polk, Daniel H.; Huang, Wen Shen;
 Reviczky, Anita; Wang, Kathy; Fisher, Delbert A.

CORPORATE SOURCE: Nucl. Med. Med. Serv., Veterans Adm. Med. Cent., Long
 Beach, CA, 90822, USA

SOURCE: American Journal of Physiology (1993), 265(1, Pt. 1),
 E115-E120

CODEN: AJPHAP; ISSN: 0002-9513

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors recently showed that thyroxine sulfate (T4S) and 3,3',5-
triiodothyronine sulfate (T3S) were major thyroid hormone metabolites in ovine
 fetuses and neonates. To further characterize the sulfation pathway in ovine
 fetuses, the authors measured 3,3',5'-*triiodothyronine* (rT3S) in serum and
 other body fluids in samples obtained from fetal (94-145 days of gestational
 age, term = 150 days), newborn and adult sheep. In addition, T3S, T4S, and
 rT3S levels were measured in tissue fluids and serum samples obtained from
 ovine fetuses 13 days after total thyroidectomy (Tx) conducted at gestational
 age of 110-113 days. Sham-operated twin fetuses served as controls. The

relative order of mean rT3S concentration for various tissue fluids in fetuses was meconium > bile > serum > allantoic fluid > urine or amniotic fluid. Peak mean tissue fluid levels generally occurred at 110-130 days gestation. In *hypothyroid* fetuses, decreases in the mean serum concns. of T4S and rT3S, but not T3S, were noted. The mean rT3S level also was decreased in allantoic fluid, bile, and meconium, whereas T4S and T3S levels were reduced only in bile of the Tx fetuses. These data demonstrate that sulfation is a major pathway in thyroid hormone metabolism in both euthyroid and *hypothyroid* ovine fetuses.

CC 2-7 (Mammalian Hormones)
ST thyroid hormone metab sulfation fetus; embryo *hypothyroid* T4 T3
rT3 sulfate
IT *Hypothyroidism*
(fetal, iodothyronine sulfate conjugates of tissues in)
IT Embryo
(thyroid hormone sulfates of, *hypothyroidism* effect on)
IT 31135-55-4 77074-49-8, Thyroxine sulfate 79349-15-8
RL: BIOL (Biological study)
(of fetal tissues in *hypothyroidism*)

L88 ANSWER 8 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 10

ACCESSION NUMBER: 1993:552539 HCAPLUS Full-text

DOCUMENT NUMBER: 119:152539

TITLE: Thyromimetic effects of 3,5,3'-
triiodothyronine sulfate in
hypothyroid rats

AUTHOR(S): Santini, Ferruccio; Hurd, Robert E.; Lee, Brenda;
Chopra, Inder J.

CORPORATE SOURCE: Cent. Health Sci., Univ. California, Los Angeles, CA,
90024-1682, USA

SOURCE: Endocrinology (1993), 133(1), 105-10
CODEN: ENDOAO; ISSN: 0013-7227

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Several parameters of the effects of thyroid hormone were examined in *hypothyroid* thyroidectomized (Tx) rats treated with T3 sulfate (T3S) or T3 [0.46 (low dose) or 2.3 (high dose) nmol/day for 10 days, i.p.]. Tx rats showed a marked degree of growth retardation, which improved after treatment with both doses of T3S and T3. The mean serum GH level was markedly reduced in Tx rats, and it improved to similar levels after treatment with the high dose of T3S and the low dose of T3. Type I monodeiodinase (MD) activity was markedly reduced in liver and kidney tissues of Tx rats. It increased in Tx rats treated with the high dose of T3S; the latter values were similar to those observed in Tx rats treated with the low dose of T3. Hepatic and renal type I MD activities increased to supranormal levels in Tx rats treated with the high dose of T3. Cardiac outer ring (5') monodeiodination of 3',5'-diiodothyronine to 3'-moniodothyronine was also reduced in Tx rats, but it improved only after treatment with the high dose of T3. Type III 5-MD activity was reduced in the cerebral cortex of Tx rats. It was restored to normal in Tx rats treated with the high dose of T3S and both doses of T3. Serum TSH, markedly elevated in Tx rats, was appreciably reduced only in rats treated with the high dose of T3. In another study, suppression of serum TSH was observed when Tx rats were treated with T3S (11.5 nmol/day) or T3 (2.3 nmol/day) for 3 days. The authors conclude that administration of T3S to *hypothyroid* rats produces thyromimetic effects, with a potency .apprx.1/5 that of T3.

CC 2-7 (Mammalian Hormones)

IT Heart, metabolism
(3',5'-diiodothyronine monodeiodination in, *triiodothyronine sulfate* regulation of)

IT Thyroid hormones
 RL: BIOL (Biological study)
 (*triiodothyronine sulfate* action as)

IT Kidney, composition
 Liver, composition
 (type I monodeiodinase of, *triiodothyronine sulfate* regulation of)

IT 4192-14-7, 3',5'-Diiodothyronine
 RL: BIOL (Biological study)
 (5'-monodeiodination of, in heart, *triiodothyronine sulfate* stimulation of)

IT 9002-71-5, Thyrotropin
 RL: PROC (Process)
 (of blood serum, *triiodothyronine sulfate* regulation of)

IT 68189-54-8
 RL: PROC (Process)
 (of cerebral cortex, *triiodothyronine sulfate* regulation of)

IT 31135-55-4
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
 (thyroid hormone-like activity of)

IT 6893-02-3, T3 Hormone
 RL: BIOL (Biological study)
 (*triiodothyronine sulfate* thyromimetic activity in relation to)

L88 ANSWER 9 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:584356 HCAPLUS Full-text

DOCUMENT NUMBER: 145:180412

TITLE: Treatment with drugs able to reduce iodine efflux significantly increases the intracellular retention time in thyroid cancer cells stably transfected with sodium iodide symporter complementary deoxyribonucleic acid

AUTHOR(S): Elisei, Rossella; Vivaldi, Agnese; Ciampi, Raffaele; Faviana, Pinuccia; Basolo, Fulvio; Santini, Ferruccio; Traino, Claudio; Pacini, Furio; Pinchera, Aldo

CORPORATE SOURCE: Department of Endocrinology and Metabolism, Section of Endocrinology, University of Pisa, Pisa, 56124, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism (2006), 91(6), 2389-2395

CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB One of the major limits of gene therapy with sodium iodide symporter (NIS), which enables cells to be subjected to radioiodine therapy, is that NIS-transfected cells rapidly release the intracellular iodine. We transfected human anaplastic (FRO) and medullary (TT) thyroid cancer-derived cell lines that were unable to take up iodine with human NIS cDNA. The possibility of increasing the iodine retention time by treating the transfected clones with myricetin, lithium, 17-(allylamino)-17-demethoxygeldanamycin (17-AAG), and 4,4'-diisothiocyantostilbene-2,2'-disulfonic acid (DIDS) was explored. We obtained 19 FRO and 16 TT clones stably transfected with NIS. Twelve of 19 FRO and nine of 16 TT clones expressed the full-length NIS mRNA; 11 of 12 FRO and four of nine TT clones were able to take up radioiodine and correctly expressed NIS protein on the plasma membrane. Kinetic anal. of iodide uptake

in the two clones (FRO-19 and TT-2) with the highest uptaking activity revealed that the plateau was reached after 30 min by FRO-19 and after 60 min by TT-2. The $t_{1/2}$ of the iodide efflux was 9 min in FRO-19 and 20 min in TT-2. The treatment of the two cell lines with four different drugs revealed that DIDS and 17-AAG, but not myricetin and lithium, significantly increased the intracellular iodide retention time in FRO-19, but not in TT-2. We showed that 17-AAG and DIDS prolong the retention time of ^{131}I in NIS-transfected thyroid tumoral cells, thus reinforcing the hope of using this approach for future clin. application, especially in patients with thyroid carcinoma who are no longer responsive to conventional therapy.

CC 1-6 (Pharmacology)

Section cross-reference(s): 3, 8

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Almeida, R	2005	15	251	Trends Cell Biol	HCAPLUS
Ambesi Impiombato, F	1980	77	3455	Proc Natl Acad Sci U	HCAPLUS
Amphoux-Fazekas, T	1998	141	129	Mol Cell Endocrinol	HCAPLUS
Armstrong, J	1992	88	105	Mol Cell Endocrinol	HCAPLUS
Arturi, F	1998	83	2193	J Clin Endocrinol Me	
Bagatell, R	2004	3	1021	Mol Cancer Ther	HCAPLUS
Braverman, L	2000			Werner and Ingbar's	
Cabantchik, Z	1992	262	C803	Am J Physiol	HCAPLUS
Cengic, N	2005	90	4457	J Clin Endocrinol Me	HCAPLUS
Cho, J	2002	9	1139	Gene Ther	HCAPLUS
Chung, J	2002	43	1188	J Nucl Med	HCAPLUS
Dai, G	1996	379	458	Nature	HCAPLUS
Devuyt, O	1997	272	C1299	Am J Physiol	HCAPLUS
Elisei, R	1994	78	867	J Clin Endocrinol Me	HCAPLUS
Elisei, R	2004	891	33	J Clin Endocrinol Me	
Elisei, R	2005	90	2403	J Clin Endocrinol Me	HCAPLUS
Fagard, M	2000	51	167	Annu Rev Plant Physi	HCAPLUS
Goddu, S	1994	35	521	J Nucl Med	MEDLINE
Haberkorn, U	2001	42	317	J Nucl Med	HCAPLUS
Haugen, B	1999	16	34	Semin Surg Oncol	MEDLINE
Huang, M	2001	8	612	Cancer Gene Ther	HCAPLUS
Jhiang, S	2000	1	205	Rev Endocr Metab Dis	HCAPLUS
Koong, S	1999	84	912	J Clin Endocrinol Me	HCAPLUS
Lee, W	2003	10	845	Oncol Rep	HCAPLUS
Marsee, D	2004	279	43990	J Biol Chem	HCAPLUS
Ong, K	1997	29	121	Gen Pharmacol	HCAPLUS
Rodriguez, A	2002	87	3500	J Clin Endocrinol Me	HCAPLUS
Rousset, B	2004			The thyroid and its	
Royaux, I	2000	141	839	Endocrinology	HCAPLUS
Santini, F	2002	87	4160	J Clin Endocrinol Me	HCAPLUS
Schlumberger, M	1999		117	Thyroid tumors	
Schroder-van der Elst,	2004	150	557	Eur J Endocrinol	
Shimura, H	1997	138	4493	Endocrinology	HCAPLUS
Smanik, P	1996	226	339	Biochem Biophys Res	HCAPLUS
Smit, J	2002	87	1247	J Clin Endocrinol Me	HCAPLUS
Smith, J	2000	10	939	Thyroid	
Spitweg, C	2000	60	6526	Cancer Res	
Spitzweg, C	1999	59	2136	Cancer Res	HCAPLUS
Spitzweg, C	2001	86	3327	J Clin Endocrinol Me	HCAPLUS
Urabe, M	1991	129	807	Endocrinology	HCAPLUS

L88 ANSWER 10 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN
 ACCESSION NUMBER: 2005:634158 HCAPLUS Full-text
 DOCUMENT NUMBER: 143:206695

TITLE: Influence of human body composition on serum peak thyrotropin (TSH) after recombinant human TSH administration in patients with differentiated thyroid carcinoma

AUTHOR(S): Castagna, Maria Grazia; *Pinchera, Aldo*; Marsili, Alessandro; Giannetti, Monica; Molinaro, Eleonora; Fierabracci, Paola; Grasso, Lucia; Pacini, Furio; *Santini, Ferruccio*; Elisei, Rossella

CORPORATE SOURCE: Department of Endocrinology and Metabolism, University of Pisa, Pisa, 56100, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism (2005), 90(7), 4047-4050
CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Objectives: In this study, we evaluated the influence of height, weight, body mass index (BMI), body surface area, and body composition [total lean body mass (LBM) and fat body mass] on serum peak TSH levels obtained after recombinant human (rh)TSH. Furthermore, to verify whether the serum peak TSH influenced the efficacy of radioiodine (¹³¹I), we compared the rate of thyroid remnant ablation according to the patients' BMI. Patients: We studied 105 patients with differentiated thyroid carcinoma who underwent rhTSH stimulation test. Serum TSH measurements were performed before and 24, 48, and 72 h after rhTSH administration. We also compared the rate of thyroid remnant ablation among 70 differentiated thyroid carcinoma patients with different BMI. Results: The serum peak TSH after rhTSH was significantly lower in overweight and obese subjects compared with normal-weight subjects (92.1±41.8, 82.4±24.2, and 112.7±46.3 µU/mL, resp.; P = 0.01) and in males compared with females (74.6±22.3 and 105.0±43.0 µU/mL, resp.; P = 0.0002). By univariate anal., serum peak TSH was neg. related to weight, height, body surface area, BMI, LBM, and fat body mass, but only LBM was independently associated with serum peak TSH levels. Although it was confirmed that overweight and obese patients had a lower serum peak TSH, the rate of ablation did not differ among normal-weight, over-weight, and obese patients. Conclusions: With this study we demonstrated that LBM is the only parameter independently associated with serum peak TSH after rhTSH administration. However, the serum peak TSH does not influence the rate of ¹³¹I remnant ablation.

CC 2-7 (Mammalian Hormones)

Section cross-reference(s): 1

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Arad, E	1993	18	662	Clin Nucl Med	MEDLINE
Barbaro, D	2003	88	4110	J Clin Endocrinol Me	HCAPLUS
de Keizer, B	2002	30	367	Eur J Nucl Med Mol I	
Haugen, B	1999	84	3877	J Clin Endocrinol Me	HCAPLUS
Ladenson, P	1997	337	888	N Engl J Med	HCAPLUS
Lippi, F	2001	144	5	Eur J Endocrinol	HCAPLUS
Logue, J	1994	67	1127	Br J Radiol	MEDLINE
Luster, M	2000	85	3640	J Clin Endocrinol Me	HCAPLUS
Mazzaferri, E	2003	88	1433	J Clin Endocrinol Me	HCAPLUS
Morgan, D	1994	26	292	Clin Pharmacokinet	HCAPLUS
Pacini, F	2002	87	4063	J Clin Endocrinol Me	HCAPLUS
Pacini, F	2004	8	21	Turk J Endocrinol Me	
Robbins, R	2001	11	865	Thyroid	MEDLINE
Roberts, C	1976	3	907	Br J Clin Pharmacol	HCAPLUS
Roubenoff, R	1991	49	163	Nutr Rev	MEDLINE
Samuel, A	1994	35	1944	J Nucl Med	MEDLINE

Sartorio, A	1988	11	727	J Endocrinol Invest	HCAPLUS
Shaikh, S	2003	57	389	Eur J Clin Nutr	MEDLINE
Thotakura, N	1991	128	341	Endocrinology	HCAPLUS
Vitale, G	2003	88	1319	J Clin Endocrinol Me	HCAPLUS

L88 ANSWER 11 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:553164 HCAPLUS Full-text

DOCUMENT NUMBER: 143:190324

TITLE: Genetic analysis of metamorphic and premetamorphic
Xenopus ciliary marginal zone

AUTHOR(S): Casarosa, S.; Leone, P.; Cannata, S.; Santini,
F.; Pinchera, A.; Barsacchi, G.;
Andreazzoli, M.

CORPORATE SOURCE: Laboratorio di Biologia Cellulare e dello Sviluppo,
Dipartimento di Fisiologia e Biochimica, Universita di
Pisa, Ghezzano-Pisa, Italy

SOURCE: Developmental Dynamics (2005), 233(2), 646-651

CODEN: DEDYEI; ISSN: 1058-8388

PUBLISHER: Wiley-Liss, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A major event affecting the eye during amphibian metamorphosis is an asym.
growth of the ventrotemporal portion of the retina compared with its
dorsonasal counterpart. This event is due to an increased proliferation of
the precursors of the ventral ciliary marginal zone (CMZ). Here, the authors
analyze the expression patterns of several key homeobox genes implicated in
eye development (Xrx1, Xvax2, Xsix3, Xpax6, Xchx10, Xotx2) to understand
whether they are active at the time in which the metamorphic changes of the
retina occur. The authors also analyze their expression patterns in the
ventral and dorsal CMZ and compare them with bromodeoxyuridine incorporation
in the CMZ. The authors' results suggest that the metamorphic CMZ maintains
the functional subdivisions described during embryonic development. Moreover,
the authors find that genes involved in proliferation and cell type
determination of the embryonic retina are actively transcribed in the
proliferating CMZ, thus indicating a potential regulatory role for these genes
in the metamorphic retina.

CC 12-3 (Nonmammalian Biochemistry)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Andreazzoli, M	2003	130	5143	Development	HCAPLUS
Barbieri, A	2002	129	805	Development	HCAPLUS
Barbieri, A	1999	96	10729	Proc Natl Acad Sci U	HCAPLUS
Belecky-Adams, T	1997	38	1293	Invest Ophthalmol Vi	MEDLINE
Bernier, G	2000	93	59	Mech Dev	HCAPLUS
Bovolenta, P	1998	70	201	Mech Dev	HCAPLUS
Burmeister, M	1996	12	376	Nat Genet	HCAPLUS
Casarosa, S	2003	22	25	Mol Cell Neurosci	HCAPLUS
Chen, C	2000	90	293	Mech Dev	MEDLINE
Chow, R	1999	126	4213	Development	HCAPLUS
Chuang, J	1999	84	195	Mech Dev	HCAPLUS
Del Bene, F	2004	427	745	Nature	HCAPLUS
Doetsch, F	1999	97	703	Cell	HCAPLUS
Dorsky, R	1995	14	487	Neuron	HCAPLUS
Furukawa, T	2000	26	383	Neuron	HCAPLUS
Ghanbari, H	2001	101	271	Mech Dev	HCAPLUS
Grant, S	1986	92	43	J Embryol Exp Morpho	MEDLINE
Heller, N	1997	69	83	Mech Dev	HCAPLUS
Hirsch, N	1997	32	45	J Neurobiol	HCAPLUS

Liu, I	1994	13	377	Neuron	HCAPLUS
Liu, Y	2001	100	115	Mech Dev	HCAPLUS
Loosli, F	2001	128	4035	Development	HCAPLUS
Loosli, F	1999	13	649	Genes Dev	HCAPLUS
Mann, F	2001	23	319	Bioessays	HCAPLUS
Marquardt, T	2001	105	43	Cell	HCAPLUS
Mathers, P	1997	387	603	Nature	HCAPLUS
Mui, S	2002	129	797	Development	HCAPLUS
Newport, J	1982	30	687	Cell	HCAPLUS
Nieuwkoop, P	1967			Normal table of deve	
Ohnuma, S	2002	129	2435	Development	HCAPLUS
Pannese, M	1995	121	707	Development	HCAPLUS
Passini, M	1997	388	495	J Comp Neurol	HCAPLUS
Perron, M	1998	199	185	Dev Biol	HCAPLUS
Straznicky, K	1971	26	67	J Embryol Exp Morpho	MEDLINE
Viczian, A	2003	130	1281	Development	HCAPLUS
Wetts, R	1989	136	254	Dev Biol	MEDLINE

L88 ANSWER 12 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:59436 HCAPLUS Full-text
TITLE: Authors' response: Haptoglobin and body mass index
AUTHOR(S): Chiellini, Chiara; Santini, Ferruccio;
Marsili, Alessandro; Vitti, Paolo; Pinchera,
Aldo; Maffei, Margherita
CORPORATE SOURCE: Dulbecco Telethon Institute, Pisa, 56126, Italy
SOURCE: Journal of Clinical Endocrinology and Metabolism
(2005), 90(1), 594-595
CODEN: JCEMAZ; ISSN: 0021-972X
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal; Letter
LANGUAGE: English

AB Unavailable

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Chiellini, C	2002	190	251	J Cell Physiol	HCAPLUS
Chiellini, C	2004	89	2678	J Clin Endocrinol Me	HCAPLUS
Cohen, P	2002	297	240	Science	HCAPLUS
De Fourmestaux, V				J Biol Chem	
Do Nascimento, C	2004	313	702	Biochem Biophys Res	
Ferrante, A	2001	50	2268	Diabetes	HCAPLUS
Friedrichs, W	1995	209	250	Bioch Biophys Res Co	HCAPLUS
Taes, Y	2005	90	594	J Clin Endocrinol Me	HCAPLUS

L88 ANSWER 13 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:59361 HCAPLUS Full-text
DOCUMENT NUMBER: 142:367991
TITLE: Lean body mass is a major determinant of levothyroxine
dosage in the treatment of thyroid diseases
AUTHOR(S): Santini, Ferruccio; Pinchera, Aldo
; Marsili, Alessandro; Ceccarini, Giovanni; Castagna,
Maria Grazia; Valeriano, Rocco; Giannetti, Monica;
Taddei, Donatella; Centoni, Roberta; Scartabelli,
Giovanna; Rago, Teresa; Mammoli, Claudia; Elisei,
Rossella; Vitti, Paolo
CORPORATE SOURCE: Department of Endocrinology, University of Pisa, Pisa,
56124, Italy
SOURCE: Journal of Clinical Endocrinology and Metabolism
(2005), 90(1), 124-127

PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Total body weight is usually employed to calculate the amount of L-T4 to be administered in patients with thyroid diseases. The aim of this study was to evaluate the effect of body composition on L-T4 requirements. Body composition was assessed by dual energy x-ray absorptiometry in 75 patients on TSH-suppressive L-T4 therapy after conventional thyroid ablation for differentiated cancer. The mean daily dose of L-T4 was lower in normal-weight (127.5 ± 21.3 $\mu\text{g/d}$) vs. overweight (139.4 ± 24.5) and obese (151.3 ± 29.1) subjects. There was a much stronger association between the L-T4 dosage and lean body mass ($P < 0.001$, $r = 0.667$) compared with fat mass ($P = 0.023$, $r = 0.26$). Measurement of regional tissue composition showed peripheral lean mass as the best correlate with the dose of L-T4 ($r = 0.679$, $P < 0.001$) whereas no correlation was observed with peripheral fat mass. In conclusion individual L-T4 requirements are dependent on lean body mass. Age-and gender-related differences in L-T4 needs reflect different proportions of lean mass over the total body weight. An estimate of lean mass may be helpful to shorten the time required to attain a stable dose of L-T4, particularly in subjects with high body mass index values that may be due either to increased muscular mass or to obesity.

CC 2-7 (Mammalian Hormones)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Alexander, E	2004	351	241	N Engl J Med	HCAPLUS
Bartalena, L	1994	21	60	Acta Med Austriaca	MEDLINE
Bracco, D	1996	81	2580	J Appl Physiol	MEDLINE
Burmeister, L	1992	75	344	J Clin Endocrinol Me	MEDLINE
Cunningham, J	1984	32	204	J Am Geriatr Soc	MEDLINE
England, M	1986	292	264	Am J Med Sci	MEDLINE
Fazio, S	2004	59	31	Recent Prog Horm Res	HCAPLUS
Fish, L	1987	316	764	N Engl J Med	MEDLINE
Franklyn, J	1994	78	1368	J Clin Endocrinol Me	MEDLINE
Friedman, J	1998	395	763	Nature	HCAPLUS
Haarbo, J	1991	11	331	Clin Physiol	MEDLINE
Hegedus, L	2003	24	102	Endocr Rev	
Hermus, A	1998	338	1438	N Engl J Med	MEDLINE
Kaplan, M	1993	93	249	Postgrad Med	MEDLINE
Leonard, J	2000		136	Werner, Ingbar's The	
Mandel, S	1993	119	492	Ann Intern Med	MEDLINE
Mazzaferri, E	1987	14	315	Semin Oncol	MEDLINE
Morgan, D	1994	26	292	Clin Pharmacokinet	HCAPLUS
Pacini, F	2002	87	1499	J Clin Endocrinol Me	HCAPLUS
Pujol, P	1996	81	4318	J Clin Endocrinol Me	HCAPLUS
Rosenbaum, R	1982	96	53	Ann Intern Med	MEDLINE
Roti, E	1993	14	401	Endocr Rev	MEDLINE
Salvatore, D	1996	137	3308	Endocrinology	HCAPLUS
Santini, F	2003	88	2825	J Clin Endocrinol Me	HCAPLUS
Sawin, C	1983	75	206	Am J Med	MEDLINE
Sawin, C	1994	331	1249	N Engl J Med	MEDLINE
Surks, M	1995	333	1688	N Engl J Med	HCAPLUS
Toft, A	1994	331	174	N Engl J Med	HCAPLUS
Uzzan, B	1996	81	4278	J Clin Endocrinol Me	HCAPLUS
Wesche, M	1998	48	701	Clin Endocrinol (Oxf)	MEDLINE
Woeber, K	2002	25	106	J Endocrinol Invest	HCAPLUS
Wolf, M	1996	134	168	Eur J Endocrinol	HCAPLUS

ACCESSION NUMBER: 2004:635974 .HCAPLUS Full-text

DOCUMENT NUMBER: 141:120896

TITLE: Serum haptoglobin: A novel marker of adiposity in humans

AUTHOR(S): Chiellini, C.; *Santini, F.*; Marsili, A.; Berti, P.; Bertacca, A.; Pelosini, C.; Scartabelli, G.; Pardini, E.; Lopez-Soriano, J.; Centoni, R.; Ciccarone, A. M.; Benzi, L.; Vitti, P.; Del Prato, S.; *Pinchera, A.*; Maffei, M.

CORPORATE SOURCE: Dulbecco Telethon Institute and Department of Endocrinology and Metabolism, Sections of Endocrinology, University of Pisa, Pisa, 56126, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism (2004), 89(6), 2678-2683

CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Haptoglobin (Hp) is a glycoprotein involved in the acute phase response to inflammation. Our previous findings indicate that Hp mRNA and protein are present in the adipose tissue of rodents and that Hp gene expression is up-regulated in obese models. The aim of the present study was to establish whether Hp could be considered a marker of obesity in humans. In 312 subjects, serum Hp was correlated directly with body mass index (BMI), leptin, C-reactive protein (CRP), and age. In a multivariate stepwise regression anal., BMI and CRP were independent determinants of serum Hp in females, with BMI having the strongest effect. CRP and age were independent determinants of serum Hp in males, although explaining only a modest percentage of the total variability. Serum Hp was pos. associated with body fat, as assessed by dual-energy x-ray absorptiometry, both in female and in male groups. The level of significance improved when serum Hp was analyzed against fat mass adjusted for lean mass. Finally, Northern and Western blot analyses performed in biopsies of s.c. abdominal fat from 20 obese individuals showed the presence of Hp mRNA and protein in the human adipose tissue. In conclusion, serum Hp constitutes a novel marker of adiposity in humans, and the adipose tissue likely contributes to determine its levels.

CC 13-6 (Mammalian Biochemistry)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abderrahim-Ferkoune, A	2003	44	994	J Lipid Res	HCAPLUS
Arredouani, M	2003	108	144	Immunology	HCAPLUS
Ausubel, F	1999			Current protocols in	
Barzilai, N	1999	892	58	Ann NY Acad Sci	MEDLINE
Bastard, J	1999	99	2221	Circulation	MEDLINE
Bensi, G	1985	4	119	EMBO J	HCAPLUS
Chehab, F	1996	12	318	Nat Genet	HCAPLUS
Chiellini, C	2002	190	251	J Cell Physiol	HCAPLUS
Chiellini, C	2003	195	309	J Cell Physiol	HCAPLUS
Das, U	2001	17	953	Nutrition	HCAPLUS
Do Nascimento, O	2004	313	702	Biochem Biophys Res	
El Ghmati, S	1996	156	2542	J Immunol	HCAPLUS
Engstrom, G	2002	22	2054	Arterioscler Thromb	
Fain, J	2004	45	536	J Lipid Res	HCAPLUS
Friedrichs, W	1995	209	250	Biochem Biophys Res	HCAPLUS
Guerre-Millo, M	2002	25	855	J Endocrinol Invest	HCAPLUS
Hannerz, J	1995	19	240	Int J Obes Relat Met	MEDLINE
Hausman, D	2001	2	239	Obes Rev	MEDLINE

Koch, W	2002	48	1377	Clin Chem	HCAPLUS
Kuchroo, V	2003	422	27	Nature	HCAPLUS
Maffei, M	1995	1	1155	Nat Med	HCAPLUS
Montani, J	2002	26	S28	Int J Obes Relat Met	HCAPLUS
Oliviero, S	1987	6	1905	EMBO J	HCAPLUS
Roubenoff, R	2003	6	295	Curr Opin Clin Nutr	
Sanna, V	2003	111	241	J Clin Invest	HCAPLUS
Taylor, S	1996	274	1151	Science	HCAPLUS
Trayhurn, P	2001	60	329	Proc Nutr Soc	HCAPLUS
Uchegbu, E	2002	25	915	J Endocrinol Invest	MEDLINE
Visser, M	1999	282	2131	JAMA	HCAPLUS
Yudkin, J	1999	19	972	Arterioscler Thromb	HCAPLUS
Zahorska-Markiewicz, B	2000	24	1392	Int J Obes Relat Met	HCAPLUS

L88 ANSWER 15 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:107562 HCAPLUS Full-text

DOCUMENT NUMBER: 143:2341

TITLE: Relative potencies and additivity of perchlorate, thiocyanate, nitrate, and iodide on the inhibition of radioactive iodide uptake by the human sodium iodide symporter

AUTHOR(S): Tonacchera, Massimo; *Pinchera, Aldo*; Dimida, Antonio; Ferrarini, Eleonora; Agretti, Patrizia; Vitti, Paolo; *Santini, Ferruccio*; Crump, Kenny; Gibbs, John

CORPORATE SOURCE: Department of Endocrinology and Metabolism and Research Center of Excellence for the Study of Damages to the Nervous and Endocrine Systems produced by Environmental, Alimentary and Pharmacological Agents, University of Pisa, Italy

SOURCE: Thyroid (2004), 14(12), 1012-1019

CODEN: THYRER; ISSN: 1050-7256

PUBLISHER: Mary Ann Liebert, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The presence of perchlorate (ClO₄⁻) in some U.S. drinking water supplies has raised concern about potential adverse thyroidal health effects, because ClO₄⁻ is known to competitively inhibit iodide uptake at the sodium iodide symporter (NIS). Humans are nutritionally and environmentally exposed to other competitive inhibitors of iodide uptake, including thiocyanate (SCN⁻) and nitrate (NO₃⁻). The joint inhibiting effects of these three anions was studied by exposing Chinese hamster ovary cells stably expressing human NIS to varying concns. of each anion sep., and in combination, and conducting measurements of ¹²⁵I- uptake. The entire data set was fit to a single Hill equation using maximum likelihood. The relative potency of ClO₄⁻ to inhibit ¹²⁵I- uptake at the NIS was found to be 15, 30 and 240 times that of SCN⁻, I⁻, and NO₃⁻ resp. on a molar concentration basis, with no evidence of synergism. These results are consistent with a common mode of action by these anions of simple competitive interaction, in which a concentration of any one of ClO₄⁻, SCN⁻, and NO₃⁻, occurring either individually or as part of a mixture of the three anions, is indistinguishable from a concentration or dilution of either one of the remaining two ions in inhibiting iodine uptake at the NIS.

CC 4-3 (Toxicology)

Section cross-reference(s): 2, 14, 61

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
=====	=====	=====	=====	=====	=====
Ajjan, R	1998	83	1217	J Clin Endocrinol Me	HCAPLUS
Alexander, W	1966	78	581	Endocrinology	HCAPLUS

Althaus, R	2001	84	1829	J Dairy Sci	HCAPLUS
Andrews, J	1973	80	810	J Obstet Gynaecol Br	MEDLINE
Banerjee, K	1997	78	679	Br J Nutr	HCAPLUS
Bottoms, S	1982	144	787	Am J Obstet Gynecol	HCAPLUS
Charmandari, E	2001	32	423	J Pediatr Gastroente	HCAPLUS
Chen, Y	1990	45	163	Arch Environ Health	HCAPLUS
Clements, F	1960	16	133	Br Med Bull	MEDLINE
Cox, D	1974			Theoretical Statisti	
Crump, C	2000	42	603	Occup Environ Med	HCAPLUS
Foss, O	1986	46	245	Scand J Clin Lab Inv	HCAPLUS
Fukata, S	1996	19	607	J Endocrinol Invest	MEDLINE
Greer, M	1966	79	237	Endocrinology	HCAPLUS
Greer, M	1962	18	187	Rec Prog Horm Res	HCAPLUS
Hauth, J	1984	63	519	Obstet Gynecol	HCAPLUS
Jo, T	1998	12	523	In Vivo	HCAPLUS
Junge, B	1985	291	22	Br Med J (Clin Res E	MEDLINE
Kassim, S	2002	35	641	Clin Biochem	HCAPLUS
Kenakin, T	1993			Analysis of Drug-Rec	
Lamm, S	1999	41	248	J Occup Environ Med	MEDLINE
Laurberg, P	2002	12	133	Thyroid	
Meberg, A	1979	68	547	Acta Paediatr Scand	HCAPLUS
Michajlovski, N	1958	312	26	Z Physiol Chem	HCAPLUS
Minamino, T	1997	17	3191	Arterioscler Thromb	MEDLINE
Moller, H	1989	18	206	Int J Epidemiol	MEDLINE
Petersen, A	1999	16	291	Food Addit Contam	HCAPLUS
Robertson, A	1987	44	351	Br J Ind Med	HCAPLUS
Rosling, H	1995	375	271	Acta Horticulturae	
Schultz, D	1985	6	847	Carcinogenesis	HCAPLUS
Schulz, V	1979	57	243	Klin Wochenschr	HCAPLUS
Soto-Blanco, B	2003	34	213	Vet Res	HCAPLUS
Taniuchi, S	2001	56	693	Allergy	HCAPLUS
Tannenbaum, S	1979	205	1332	Science	MEDLINE
Tonacchera, M	2001	144	611	Eur J Endocrinol	HCAPLUS
Urbansky, E	2002	9	187	Environ Sci Pollut R	HCAPLUS
Vanderpas, J	1984	20	327	Clin Endocrinol	MEDLINE
Vanetten, C	1969		103	Goitrogens	HCAPLUS
Venzon, D	1988	37	87	Appl Stat	
Virtanen, A	1963	3	23	Zeit Ernahrungswisse	
Wagner, D	1984	57	247	IARC Sci Publ	HCAPLUS
Watanabe, T	2000	301	169	Clin Chim Acta	HCAPLUS
Wyngaarden, J	1952	50	537	Endocrinology	HCAPLUS
Wyngaarden, J.	1953	52	568	Endocrinology	HCAPLUS
Ysart, G	1999	16	521	Food Addit Contam	HCAPLUS
Zapico, P	1991	74	783	J Dairy Sci	HCAPLUS

L88 ANSWER 16 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:157063 HCAPLUS Full-text

DOCUMENT NUMBER: 140:268997

TITLE: Genetic screening for melanocortin-4 receptor mutations in a cohort of italian obese patients: Description and functional characterization of a novel mutation

AUTHOR(S): **Santini, Ferruccio**; Maffei, Margherita; Ceccarini, Giovanni; Pelosini, Caterina; Scartabelli, Giovanna; Rosellini, Veronica; Chiellini, Chiara; Marsili, Alessandro; Lisi, Simonetta; Tonacchera, Massimo; Agretti, Patrizia; Chiovato, Luca; Mammoli, Claudia; Vitti, Paolo; **Pinchera, Aldo**

CORPORATE SOURCE: Department of Endocrinology and Metabolism, University of Pisa, Pisa, 56124, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism
(2004), 89(2), 904-908
CODEN: JCEMAZ; ISSN: 0021-972X
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mutations in the human melanocortin-4 receptor (MC4-R) gene may account for up to 5.8% of morbid nonsyndromic obesity. We have screened 120 unrelated obese patients for variants of the MC4-R gene. Four heterozygous missense variants were detected, including two polymorphisms (Val103Ile and Ile251Leu) previously described in the literature. A novel heterozygous mutation (Glu308Lys) was detected in a 36-yr-old female patient. Compared with the wild-type receptor, cells expressing the mutated receptor showed a reduced stimulation of cAMP production and a reduction of radioactive α MSH binding. No segregation of the mutation with the obese phenotype could be demonstrated. A second, potentially pathogenic mutation (Ser30Phe) was detected in a 31-yr-old female patient. Functional anal. of the mutated receptor showed no change in the affinity to the natural ligand α MSH nor limited ability to stimulate cAMP production. Sixty lean subjects were also screened, and no addnl. variants of the MC4-R gene were observed, except for two individuals with the Val103Ile polymorphism. In conclusion, we have screened a population of Italian obese subjects for MC4-R variants, demonstrating a 1.7% prevalence of potentially pathogenic mutations. A novel heterozygous missense mutation (Glu308Lys) that impairs MC4-R functional activity in vitro was characterized.

CC 14-14 (Mammalian Pathological Biochemistry)

Section cross-reference(s): 2, 3

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Arch, J	2002	25	867	J Endocrinol Invest	HCAPLUS
Chiovato, L	1987	10	383	J Endocrinol Invest	MEDLINE
Cone, R	2000	106	185	J Clin Invest	HCAPLUS
Cone, R	1999	10	211	Trends Endocrinol Me	HCAPLUS
Cummings, D	2003	54	453	Annu Rev Med	HCAPLUS
Dubern, B	2001	139	204	J Pediatr	HCAPLUS
Farooqi, I	2000	83	31	Arch Dis Child	MEDLINE
Farooqi, I	2000	106	271	J Clin Invest	HCAPLUS
Farooqi, I	2003	348	1085	N Engl J Med	HCAPLUS
Friedman, J	2003	299	856	Science	HCAPLUS
Gu, W	1999	48	635	Diabetes	HCAPLUS
Hinney, A	1999	84	1483	J Clin Endocrinol Me	HCAPLUS
Ho, G	1999	274	35816	J Biol Chem	HCAPLUS
Jacobson, P	2002	87	4442	J Clin Endocrinol Me	HCAPLUS
Lopata, M	1984	12	5707	Nucleic Acids Res	HCAPLUS
Miraglia Del Giudice, E	2002	26	647	Int J Obes Relat Met	HCAPLUS
Mountjoy, K	1992	257	1248	Science	HCAPLUS
Vaisse, C	2000	106	253	J Clin Invest	HCAPLUS
Vaisse, C	1998	20	113	Nat Genet	HCAPLUS
Vettor, R	2002	25	836	J Endocrinol Invest	HCAPLUS
Yeo, G	2003	12	561	Hum Mol Genet	HCAPLUS
Yeo, G	1998	20	111	Nat Genet	HCAPLUS

L88 ANSWER 17 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STM

ACCESSION NUMBER: 2004:478218 HCAPLUS Full-text

DOCUMENT NUMBER: 141:138292

TITLE: Serum concentrations of adiponectin and leptin in patients with thyroid dysfunctions

AUTHOR(S): Santini, F.; Marsili, A.; Mammoli, C.; Valeriano, R.; Scartabelli, G.; Pelosini, C.;

Giannetti, M.; Centoni, R.; Vitti, P.; *Pinchera, A.*

CORPORATE SOURCE: Department of Endocrinology, University of Pisa, Pisa, Italy

SOURCE: Journal of Endocrinological Investigation (2004), 27(2), RC5-RC7

CODEN: JEIND7; ISSN: 0391-4097

PUBLISHER: Editrice Kurtis s.r.l.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Thyroid dysfunction is associated with metabolic changes that affect mass and adipocyte function, as well as lipid and carbohydrate metabolism. Adipose tissue performs complex metabolic and endocrine functions. Leptin and adiponectin are two of the most important adipocytokines, both involved in the regulation of intermediate metabolism. The aim of this study was to evaluate the relationships between thyroid status and circulating levels of the two adipose tissue hormones. We studied 15 patients with hyperthyroidism, 15 patients with hypothyroidism and 15 euthyroid subjects, all matched by sex, age and body mass index (BMI). Serum concns. of free thyroxine, free triiodothyronine, TSH, leptin and adiponectin and anthropometric parameters (weight, height, BMI) were assessed. No significant difference was found among the 3 groups, as assessed by Student's t-test, both for adiponectin and leptin. We conclude that metabolic changes associated with thyroid dysfunction are not related to variations in serum levels of adiponectin or leptin.

CC 14-8 (Mammalian Pathological Biochemistry)

Section cross-reference(s): 2

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Arita, Y	1999	257	79	Biochem Biophys Res	HCAPLUS
Berg, A	2002	13	84	Trends Endocrinol Me	HCAPLUS
Biondi, B	2002	12	505	Thyroid	
Bruun, J	2003	285	527	Am J Physiol Endocri	
Corbetta, S	1997	137	659	Eur J Endocrinol	HCAPLUS
Delporte, M	2002	367	677	Biochem J	HCAPLUS
Dimitriadis, G	2001	109	225	Exp Clin Endocrinol	
Duntas, L	2002	12	287	Thyroid	HCAPLUS
Fasshauer, M	2002	290	1084	Biochem Biophys Res	HCAPLUS
Fernandez-Real, J	2003	88	2714	J Clin Endocrinol Me	HCAPLUS
Guerre-Millo, M	2002	25	855	J Endocrinol Invest	HCAPLUS
Hsueh, W	2003	92	3	Am J Cardiol	
Iglesias, P	2003	59	621	Clin Endocrinol (Oxf	HCAPLUS
Ineck, B	2003	37	725	Ann Pharmacother	HCAPLUS
Kautzky-Willer, A	1999	29	395	Eur J Clin Invest	HCAPLUS
Korbonits, M	1998	49	569	Clin Endocrinol (Oxf	MEDLINE
Kubota, N	2002	277	25863	J Biol Chem	HCAPLUS
Lindsay, R	2002	360	57	Lancet	HCAPLUS
Matsubara, M	2000	7	50	J Atheroscler Thromb	HCAPLUS
Matsuda, M	2002	277	37487	J Biol Chem	HCAPLUS
Prins, J	2000	16	639	Best Pract Res Clin	
Pucci, E	2000	24	109	Int J Obes Relat Met	
Ravussin, E	2002	967	363	Ann N Y Acad Sci	HCAPLUS
Sreenan, S	1997	7	407	Thyroid	MEDLINE
Tsao, T	2002	440	213	Eur J Pharmacol	HCAPLUS
Ukkola, O	2002	80	696	J Mol Med	HCAPLUS
Weyer, C	2001	86	1930	J Clin Endocrinol Me	HCAPLUS
Yamamoto, Y	2002	103	137	Clin Sci (Lond)	HCAPLUS
Yamauchi, T	2001	7	941	Nat Med	HCAPLUS

Yamauchi, T	2003	423	762	Nature	HCAPLUS
Yang, W	2001	86	3815	J Clin Endocrinol Me	HCAPLUS
Zhang, Y	2002	1584	115	Biochim Biophys Acta	HCAPLUS

L88 ANSWER 18 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:474833 HCAPLUS Full-text

DOCUMENT NUMBER: 139:208054

TITLE: Role for inner ring deiodination preventing transcutaneous passage of thyroxine

AUTHOR(S): *Santini, Ferruccio*; Vitti, Paolo; Chiovato, Luca; Ceccarini, Giovanni; Macchia, Marco; Montanelli, Lucia; Gatti, Gianluca; Rosellini, Veronica; Mammoli, Claudia; Martino, Enio; Chopra, Inder J.; Safer, Joshua D.; Braverman, Lewis E.; *Pinchera, Aldo*

CORPORATE SOURCE: Department of Endocrinology, University of Pisa, Pisa, 56124, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism (2003), 88(6), 2825-2830

CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Creams containing thyroid hormone are commonly employed for cosmetic purposes. To verify whether T4 applied to the skin surface can enter the bloodstream either directly or as a metabolite, a cream containing L-T4 [3,5,3',5'-tetraiodothyronine (T4)] was self-applied by volunteers for 2 wk. No significant variations in urinary iodide, TSH, and serum (total and free) T4 and T3 concns. were observed at any time relative to pretreatment values, whereas rT3 concns. increased significantly 6 and 12 h after cream application. The increased rT3 concentration led the authors to investigate the presence of inner ring type III deiodinase (D3) activity in human skin. Using human surgical discard skin, the authors found that T4 can be carried across human epidermis in a liposome cream. Substantial inner ring deiodination was suggested by the fact that only 10% of transferred thyroid hormone remained as T4, and T3 was not detected. The authors then measured D3 activity in a surgical skin specimen. The Km for T3 was 1.74 nmol/L, and the maximum velocity was 23.5 fmol/μg microsomal protein/h. In conclusion, the authors' study indicates that normal human skin serves as a substantial, but incomplete, barrier to T4 passage. D3 plays an important role in augmenting T4 blockade by inactivating T4 to rT3.

CC 2-7 (Mammalian Hormones)

Section cross-reference(s): 63

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Archer, C	1998		113	Rook, Wilkinson, and	
Bashir, S	2001	7	40	Skin Res Technol	MEDLINE
Bates, J	1999	140	844	Endocrinology	HCAPLUS
Becker, K	1997	138	2989	Endocrinology	HCAPLUS
Bernal, J	2002	25	268	J Endocrinol Invest	MEDLINE
Bianco, A	2002	23	38	Endocr Rev	HCAPLUS
Burrow, G	1994	20	1072	N Engl J Med	
Calvo, R	1998	801	150	Brain Res	HCAPLUS
Campos-Barros, A	1996	81	2179	J Clin Endocrinol Me	HCAPLUS
Castro, M	1985	76	1921	J Clin Invest	HCAPLUS
Chopra, I	2000		121	Werner, Ingbar's the	
Del Guerra, P	1992	63	373	Int Arch Occup Envir	MEDLINE
Fleisher, D	1995	57	1293	Life Sci	HCAPLUS
Germain, D	1997	7	655	Thyroid	

Hennemann, G	2001	22	451	Endocr Rev	HCAPLUS
Huang, T	1988	23	196	Pediatr Res	HCAPLUS
Kao, J	1990	22	363	Drug Metab Rev	HCAPLUS
Leonard, J	2000		136	Werner, Ingbar's the	
Rapaka, R	1981	70	131	J Pharm Sci	HCAPLUS
Redelmeier, T	1999		2699	Fitzpatrick's dermat	
Rendl, J	1998	106	S34	Exp Clin Endocrinol	
Richard, K	1998	83	2868	J Clin Endocrinol Me	HCAPLUS
Riviere, J	1992	81	601	J Pharm Sci	HCAPLUS
Roti, E	1981	53	498	J Clin Endocrinol Me	HCAPLUS
Roti, E	1981	94	183	Trans Assoc Am Physi	HCAPLUS
Safer, J	2001	11	717	Thyroid	HCAPLUS
Salvatore, D	1995	96	2421	J Clin Invest	HCAPLUS
Santini, F	1992	130	2325	Endocrinology	HCAPLUS
Santini, F	2001	144	577	Eur J Endocrinol	HCAPLUS
Santini, F	1992	74	1366	J Clin Endocrinol Me	HCAPLUS
Santini, F	1999	84	493	J Clin Endocrinol Me	HCAPLUS
Villar, D	2000	68	119	Res Vet Sci	HCAPLUS

L88 ANSWER 19 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:255396 HCAPLUS Full-text

DOCUMENT NUMBER: 141:289209

TITLE: In vitro assay of thyroid disruptors affecting TSH-stimulated adenylate cyclase activity

AUTHOR(S): Santini, F.; Vitti, P.; Ceccarini, G.; Mammoli, C.; Rosellini, V.; Pelosini, C.; Marsili, A.; Tonacchera, M.; Agretti, P.; Santoni, T.; Chiovato, L.; Pinchera, A.

CORPORATE SOURCE: Department of Endocrinology, Centro di Eccellenza AmbISEN, University of Pisa, Pisa, Italy

SOURCE: Journal of Endocrinological Investigation (2003), 26(10), 950-955

CODEN: JEIND7; ISSN: 0391-4097

PUBLISHER: Editrice Kurtis s.r.l.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Several natural or synthetic chems. have been indicated as potential thyroid disruptors. The development of in vitro assays has been recommended to comprehensively assess the potential thyroid disrupting activity of a substance or a complex mixture. In this study, 12 substances suspected for acting as thyroid disruptors were tested for their ability to inhibit TSH-stimulated cAMP production in vitro. Those substances producing an inhibition were further studied to establish the level at which they interfere with this step of thyroid cell function. Using Chinese hamster ovary cells (CHO) transfected with the recombinant human TSH receptor, a dose-dependent inhibition of TSH-stimulated adenylate cyclase activity was produced by 1,1-bis-(4-chlorophenyl)-2,2,2-trichloroethane (DDT), Aroclor 1254 and Melissa officinalis. All three substances also inhibited the cAMP production stimulated by TSH receptor antibody. Melissa officinalis produced a significant inhibition of TSH binding to its receptor and of antibody binding to TSH, while no significant changes were produced by Aroclor 1254 or DDT in these assays. These data suggest that principles contained in Melissa officinalis may block the binding of TSH to its receptor by acting both on the hormone and the receptor itself, while DDT and Aroclor 1254 affect cAMP production mainly at post-receptor step. In conclusion, the authors have developed a set of in vitro assays that allow investigation into the effect of thyroid disruptors on the TSH-mediated activation of the cAMP cascade. These assays may be useful to identify the mechanism of action of thyroid disruptors, coming beside and supporting animal studies or epidemiol. surveys.

CC 2-1 (Mammalian Hormones)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
=====	=====	=====	=====	=====	=====
Auf'mkolk, M	1984	115	527	Endocrinology	HCAPLUS
Bernal, J	2002	25	268	J Endocrinol Invest	MEDLINE
Bigsby, R	1999	4	613	Environ Health Persp	
Brucker-Davis, F	1998	8	827	Thyroid	MEDLINE
Byrne, J	1987	121	520	Endocrinology	HCAPLUS
Chiovato, L	1994	17	809	J Endocrinol Invest	HCAPLUS
Colborn, T	2002	3	363	Environ Health Persp	
De Vito, M	1999	107	407	Environ Health Persp	MEDLINE
Gaitan, E	2000		69	Merk European Thyroi	
Howdeshell, K	2002	3	337	Environ Health Persp	
Jefferies, D	1969	166	1278	Science	HCAPLUS
Kohrle, J	2000		41	Merk European Thyroi	
Koopman-Esseboom, C	1994	36	468	Pediatr Res	MEDLINE
Leatherland, J	1998	14	41	Toxicol Ind Health	HCAPLUS
Longnecker, M	2000	11	249	Epidemiology	MEDLINE
Marino, M	2000	279	1295	Am J Physiol Cell Ph	
Matsuura, N	2001	45	1167	Chemosphere	HCAPLUS
Moccia, R	1977	198	425	Science	HCAPLUS
Osius, N	1999	107	843	Environ Health Persp	HCAPLUS
Perret, J	1990	171	1044	Biochem Biophys Res	HCAPLUS
Persky, V	2001	109	1275	Environ Health Persp	HCAPLUS
Porterfield, S	2000	3	433	Environ Health Persp	
Sandau, C	2002	110	411	Environ Health Persp	HCAPLUS
Santini, F	1995	110	195	Mol Cell Endocrinol	HCAPLUS
Schumacher, U	1993	29	103	J Wildl Dis	MEDLINE
Tonacchera, M	1996	44	621	Clin Endocrinol (Oxf	HCAPLUS
Turusov, V	2002	110	125	Environ Health Persp	HCAPLUS
Vitti, P	1982	5	179	J Endocrinol Invest	HCAPLUS
Zacharewski, T	1998	2	577	Environ Health Persp	
Zoeller, T	2002	3	355	Environ Health Persp	

L88 ANSWER 20 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:466632 HCAPLUS Full-text

DOCUMENT NUMBER: 137:16060

TITLE: Non-RIA methods for diagnosing thyroid conditions and
for monitoring thyroxine therapy by analyzing TSH and
thyroid hormones in urine

INVENTOR(S): Salhanick, Hilton A.; Hourihan, Joachim

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 32 pp., Cont.-in-part of U.S.
Provisional Ser. No. 220,894.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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US 2002076827	A1	20020620	US 2001-915931	20010726
PRIORITY APPLN. INFO.:			US 2000-220894P	P 20000726

AB This invention provides a method of diagnosing a thyroid condition in a subject which comprises: determining the concentration of TSH in a urine sample by a method which is not a RIA; and comparing the concentration of TSH with a urinary concentration of TSH in a normal subject; wherein: (i) a concentration of TSH which is higher than the urinary concentration of TSH in

the normal subject diagnoses *hypothyroidism* in the subject; and (ii) a concentration of TSH which is lower than the urinary concentration of TSH in the normal subject diagnoses hyperthyroidism in the subject. The method used for the determination comprises: (1) contacting an agent capable of binding to TSH with the urine sample so as to bind TSH which is present in the sample to the agent; (2) removing unbound urine sample; (3) contacting the bound TSH with a detectable agent capable of binding to TSH so as to bind the detectable agent to the bound TSH; (4) removing unbound detectable agent; and (5) determining the amount of detectable agent which is bound to the TSH, thereby determining the amount of TSH in the urine sample. The binding agent is either an antibody or a receptor and can be immobilized on a gold particle, a latex particle, a magnetic particle or other solid phase. The detectable agent is a marker which is more specifically a colorimetric, a luminescent, or a fluorescent marker. Instead of TSH, triiodothyronine, *triiodothyronine sulfate*, thyroxine, or thyroxine glucuronide can be determined. This invention also proves a method of monitoring thyroxine therapy.

IC ICM G01N033-566

ICS G01N033-543

INCL 436501000

CC 2-1 (Mammalian Hormones)

IT Diagnosis

Hyperthyroidism

Hypothyroidism

Thyroid gland, disease

Urine analysis

(non-RIA methods for diagnosing *thyroid* conditions and for monitoring thyroxine therapy by analyzing TSH and *thyroid* hormones in urine)

IT 6893-02-3, Triiodothyronine 9002-71-5, TSH 21462-56-6, Thyroxine glucuronide 31135-55-4, *Triiodothyronine sulfate*

RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)

(non-RIA methods for diagnosing thyroid conditions and for monitoring thyroxine therapy by analyzing TSH and thyroid hormones in urine)

L88 ANSWER 21 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:717606 HCAPLUS Full-text

DOCUMENT NUMBER: 137:257342

TITLE: Cytotoxic effects of carboplatinum and epirubicin in the setting of an elevated serum thyrotropin for advanced poorly differentiated thyroid cancer

AUTHOR(S): *Santini, Ferruccio*; Bottici, Valeria; Elisei, Rossella; Montanelli, Lucia; Mazzeo, Salvatore; Basolo, Fulvio; *Pinchera, Aldo*; Pacini, Furio

CORPORATE SOURCE: Department of Endocrinology, University of Pisa, Pisa, 56124, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism (2002), 87(9), 4160-4165
CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Chemotherapy represents the only therapeutic option in most poorly differentiated thyroid carcinomas, although its effect is limited and short lasting. The aim of this study was to evaluate whether increasing the metabolic rate of thyroid cancer cells by TSH stimulation might result in higher response rate to chemotherapy. Fourteen patients with poorly differentiated thyroid carcinoma and nonfunctioning diffuse lung metastases

detected at computed tomog. scan, entered this study. A combination of carboplatinum and epirubicin was administered at 4- to 6-wk intervals for six courses. TSH stimulation was achieved by reduction of the daily dose of L-thyroxine resulting in mild hypothyroidism (eight patients) or by administration of recombinant human TSH (six patients). Two addnl. patients did not complete the therapeutic protocol due to severe hematol. side effects. Results were evaluated by comparison of lung computed tomog. scans before and after therapy. One patient had a complete remission. Five patients had partial remission, and seven patients had disease stabilization. One patient progressed to death. The overall rate of pos. responses was 37% that rose to 81% when including patients with stable disease. Serum thyroglobulin after chemotherapy declined more than 50% in six patients, with respect to basal levels. Apparently, no difference in the response rate was observed between exogenous or endogenous TSH stimulation. At the time of this anal., among the patients who completed the treatment courses, 9 of 14 patients (64.3%) are still alive (median survival from start of chemotherapy = 21 mo, range: 15-34). Six of these patients did not show progression of lung disease, whereas regrowth of lung metastases was observed in three patients after 19, 20, and 21 mo from chemotherapy, resp. Five patients died of their disease, including the one who had progression of lung disease during chemotherapy, three who died for brain or bone metastases, and one who died for refractory local tumor invasion. No progression of lung metastases was observed until death in these four patients. In conclusion, the response rate of poorly differentiated thyroid cancer to chemotherapy observed in this study was favorable and promising. TSH stimulation may have contributed to these results.

CC 1-6 (Pharmacology)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
=====					
Ahuja, S	1987	10	303	J Endocrinol Invest	MEDLINE
Blum, R	1996		37	Current cancer thera	
Carayon, P	1980	51	915	J Clin Endocrinol Me	MEDLINE
Casara, D	1993	34	1626	J Nucl Med	MEDLINE
De Besi, P	1991	14	475	J Endocrinol Invest	MEDLINE
Haugen, B	1999	84	3877	J Clin Endocrinol Me	HCAPLUS
Hoskin, P	1987	10	187	Radiother Oncol	MEDLINE
Mazzaferri, E	1994	97	418	Am J Med	MEDLINE
Morris, J	1997	7	63	Thyroid	MEDLINE
Pacini, F	1999	81	463	Biochimie	HCAPLUS
Pacini, F	1984	10	911	Drugs Exp Clin Res	
Pacini, F	2001	86	4092	J Clin Endocrinol Me	HCAPLUS
Pacini, F	1987	28	1888	J Nucl Med	MEDLINE
Pacini, F	1994	1	921	Oncol Rep	
Pacini, F	1994	18	600	World J Surg	MEDLINE
Pineda, J	1995	80	1488	J Clin Endocrinol Me	HCAPLUS
Pollina, L	1996	73	139	Br J Cancer	MEDLINE
Schlumberger, M	1980	51	513	J Clin Endocrinol Me	MEDLINE
Schlumberger, M	1999	22	3	J Endocrinol Invest	
Schlumberger, M	1999	22	35	J Endocrinol Invest	
Schlumberger, M	1988	29	4790	J Nucl Med	
Schlumberger, M	1996	37	598	J Nucl Med	HCAPLUS
Schlumberger, M	1998	338	297	N Engl J Med	MEDLINE
Schlumberger, M	1999			Thyroid tumors	
Shimaoka, K	1985	56	2155	Cancer	MEDLINE

L88 ANSWER 22 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:108457 HCAPLUS Full-text

DOCUMENT NUMBER: 137:16734

TITLE: Effect of microsomal enzyme inducers on the biliary

excretion of triiodothyronine (T3) and its metabolites
 AUTHOR(S): Vansell, Nichole R.; Klaassen, Curtis D.
 CORPORATE SOURCE: Department of Pharmacology, Toxicology, University of
 Kansas Medical Center, Kansas City, KS, 66160-7417,
 USA
 SOURCE: Toxicological Sciences (2002), 65(2), 184-191
 CODEN: TOSCF2; ISSN: 1096-6080
 PUBLISHER: Oxford University Press
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB It has been postulated that inducers of UDP-glucuronosyltransferase (UGT) decrease circulating thyroid hormone concns. by increasing their biliary excretion. The inducers pregnenolone-16 α -carbonitrile (PCN), 3-methylcholanthrene (3MC), and Aroclor 1254 (PCB) are each effective at reducing serum thyroxine concns. However, only PCN treatment produces a marked increase in serum levels of TSH, whereas 3MC and PCB cause little to no increase in TSH. Excessive TSH elevation is considered the primary stimulus for thyroid tumor development in rats, yet the mechanism by which enzyme induction leads to TSH elevation is not fully understood. Whereas PCN, 3MC, and PCB all increase microsomal UGT activity toward T4, only PCN causes an increase in T3-UGT activity in vitro. The purpose of this study was to determine whether PCN, which increases serum TSH, causes an increase in the glucuronidation and biliary excretion of T3 in vivo. Male rats were fed control diet or diet containing PCN (1000 ppm), 3MC (250 ppm), or PCB (100 ppm) for 7 days. Animals were then given [125I]-T3, iv, and bile was collected for 2 h. Radiolabeled metabolites in bile were analyzed by reverse-phase HPLC with γ -detection. The biliary excretion of total radioactivity was increased up to 75% by PCN, but not by 3MC or PCB. Of the T3 excreted into bile, approx. 75% was recovered as T3-glucuronide, with remaining amts. represented as T3-sulfate, T2-sulfate, T3, and T2. Biliary excretion of T3-glucuronide was increased up to 66% by PCN, while neither 3MC nor PCB altered T3-glucuronide excretion. These findings indicate that PCN increases the glucuronidation and biliary excretion of T3 in vivo, and suggest that enhanced elimination of T3 may be the mechanism responsible for the increases in serum TSH caused by PCN.

CC 4-6 (Toxicology)

IT Bile
 Carcinogens
 Glucuronylation
 Microsome

Thyroid gland, neoplasm

(effect of microsomal enzyme inducers on biliary excretion of triiodothyronine and metabolites)

IT 51-48-9, Thyroxine, biological studies 1041-01-6, 3,5-Diiodo-L-thyronine
 6893-02-3, Triiodothyronine 6893-02-3D, Triiodothyronine, metabolites
 9002-71-5, TSH 9030-08-4, UDP-glucuronosyltransferase 29919-72-0,
 Triiodothyronine glucuronide 31135-55-4,
 Triiodothyronine sulfate 64192-57-0,
 3,3'-Diiodo-L-thyronine sulfate

RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (effect of microsomal enzyme inducers on biliary excretion of triiodothyronine and metabolites)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abe, M	1998	174	186	J Cell Physiol	HCAPLUS
Arand, M	1991	77	97	Chem Biol Interact	HCAPLUS
Barter, R	1992	113	36	Toxicol Appl Pharmac	HCAPLUS
Barter, R	1994	128	9	Toxicol Appl Pharmac	HCAPLUS

Bastomsky, C	1976	98	1309	Endocrinology	HCAPLUS
Bastomsky, C	1977	101	292	Endocrinology	HCAPLUS
Bastomsky, C	1973	56	267	J Endocrinol	HCAPLUS
Beetstra, J	1991	128	741	Endocrinology	HCAPLUS
Comer, C	1985	80	427	Toxicol Appl Pharmac	HCAPLUS
Connell, J	1984	26	453	Eur J Clin Pharmacol	HCAPLUS
Curran, P	1991	12	135	Endocr Rev	HCAPLUS
de Herder, W	1988	122	153	Endocrinology	HCAPLUS
Docter, R	1997	138	1841	Endocrinology	HCAPLUS
Dunn, R	1999	290	319	J Pharmacol Exp Ther	HCAPLUS
Eelkman Rooda, S	1989	124	740	Endocrinology	
Emi, Y	1995	117	392	J Biochem (Tokyo)	HCAPLUS
Fournel, S	1987	17	445	Xenobiotica	MEDLINE
Fujita, K	1999	79	467	Jpn J Pharmacol	HCAPLUS
Gorski, J	1987	44	297	Toxicology	HCAPLUS
Haque, S	1991	10	515	DNA Cell Biol	HCAPLUS
Hennemann, G	1998	48	1	Clin Endocrinol (Oxf)	HCAPLUS
Hennemann, G	1997		75	Pharmacotherapeutics	HCAPLUS
Henry, E	1987	89	165	Toxicol Appl Pharmac	HCAPLUS
Hood, A	1999	160	163	Toxicol Appl Pharmac	HCAPLUS
Hood, A	2000	163	240	Toxicol Appl Pharmac	HCAPLUS
Hood, A	1999	50	45	Toxicol Sci	HCAPLUS
Hood, A	2000	55	78	Toxicol Sci	HCAPLUS
Jones, H	1993	67	622	Arch Toxicol	HCAPLUS
Kanno, J	1990	18	239	Toxicol Pathol	HCAPLUS
Lilienblum, W	1982	31	907	Biochem Pharmacol	HCAPLUS
Liu, J	1995	273	977	J Pharmacol Exp Ther	HCAPLUS
Lu, R	1996	270	F332	Am J Physiol	HCAPLUS
Masubuchi, N	1997	54	1225	Biochem Pharmacol	HCAPLUS
McClain, R	1988	94	254	Toxicol Appl Pharmac	HCAPLUS
McClain, R	1989	99	216	Toxicol Appl Pharmac	HCAPLUS
Mendoza, D	1966	79	106	Endocrinology	HCAPLUS
Moreno, M	1994	344	143	FEBS Lett	HCAPLUS
Rausch-Derra, L	2001	33	1469	Hepatology	HCAPLUS
Rondeel, J	1995	61	421	Neuroendocrinology	HCAPLUS
Rooda, S	1989	125	2187	Endocrinology	MEDLINE
Runge-Morris, M	1998	109	315	Chem Biol Interact	HCAPLUS
Rutgers, M	1989	125	2175	Endocrinology	HCAPLUS
Schuur, A	1997	138	3727	Endocrinology	HCAPLUS
Semler, D	1989	98	263	Toxicol Appl Pharmac	HCAPLUS
Sonderfan, A	1988	265	208	Arch Biochem Biophys	HCAPLUS
Ullrich, D	1984	33	97	Biochem Pharmacol	HCAPLUS
van Raaij, J	1993	45	627	Biochem Pharmacol	HCAPLUS
Visser, T	1992	19	18	Acta Med Austriaca	
Visser, T	1996	23	10	Acta Med Austriaca	MEDLINE
Visser, T	1991	42	444	Biochem Pharmacol	HCAPLUS
Visser, T	1998	109	279	Chem Biol Interact	HCAPLUS
Visser, T	1983	112	1547	Endocrinology	HCAPLUS
Visser, T	1993	133	2177	Endocrinology	HCAPLUS
Visser, T	1993	315	65	FEBS Lett	HCAPLUS
Visser, T	1984	14	35	Horm Metab Res Suppl	HCAPLUS
Visser, T	1990		255	The Thyroid Gland	
Watkins, J	1982	64	439	Toxicol Appl Pharmac	HCAPLUS
Wilson, A	1996	33	16	Fundam Appl Toxicol	HCAPLUS

L88 ANSWER 23 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:310978 HCAPLUS Full-text

DOCUMENT NUMBER: 136:379875

TITLE: Iopanoic acid rapidly controls type I
amiodarone-induced thyrotoxicosis prior to

thyroidectomy

AUTHOR(S): Bogazzi, F.; Aghini-Lombardi, F.; Cosci, C.; Lupi, I.; Santini, F.; Tanda, M. L.; Miccoli, P.; Basolo, F.; Pinchera, A.; Bartalena, L.; Braverman, L. E.; Martino, E.

CORPORATE SOURCE: Department of Endocrinology and Metabolism, Boston Medical Center, Boston, MA, USA

SOURCE: Journal of Endocrinological Investigation (2002), 25(2), 176-180
CODEN: JEIND7; ISSN: 0391-4097

PUBLISHER: Editrice Kurtis s.r.l.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Amiodarone-induced thyrotoxicosis (AIT) may develop either in apparently normal thyroid glands (Type II AIT) or in the presence of sub-clin. thyroid abnormalities (either autonomous goiter or latent Graves' disease; Type I AIT). Mixed forms also occur. While Type I AIT is due to iodine-induced excess thyroid hormone synthesis, Type II AIT is a form of amiodarone (possibly iodine) -induced destructive thyroiditis. Type I AIT is usually treated by combined thion-amide and potassium perchlorate therapy, but may be resistant to therapy. On the other hand, Type II AIT often responds favorably to gluco-corticoids and may not require further therapy once euthyroidism has been restored. Not infrequently, however, AIT (especially Type I) is resistant to conventional treatment, and several weeks or months may elapse before euthyroidism is restored. Thyroidectomy has been carried out in Type I AIT patients, but thyroid surgery in thyrotoxic patients, especially those with underlying cardiac problems, carries a high surgical risk. In this study we describe 3 patients with Type I AIT, who were successfully treated with a short course of iopanoic acid (IOP), an oral cholecystog. agent, which is rich in iodine and is a potent inhibitor of 5'-deiodinase, resulting in a marked decrease in the peripheral tissue conversion of T4 to T3, in preparation for thyroid surgery. Euthyroidism was rapidly restored in 7-12 days, allowing a subsequent safe and uneventful thyroidectomy in all cases. These patients were then treated with L-T4 for their hypothyroidism and amiodarone was safely re-instituted. We suggest that IOP is the drug of choice in the rapid restoration of euthyroidism prior to definitive thyroidectomy in patients with drug resistant Type I AIT.

CC 1-10 (Pharmacology)
Section cross-reference(s): 8

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Aghini-Lombardi, F	1993	16	823	J Endocrinol Invest	MEDLINE
Baeza, A	1991	35	439	Clin Endocrinol	MEDLINE
Bartalena, L	2001	81	2930	J Clin Endocrinol Met	
Bartalena, L	2001	4	116	J Endocrinol Invest	
Bogazzi, F	1997	7	541	Thyroid	MEDLINE
Braga, M	2001	86	1853	J Clin Endocrinol Me	HCAPLUS
Brennan, M	1987	102	1062	Surgery	MEDLINE
Claxton, S	2000	70	155	Aust NZJ Surg	
Daniels, G	2001	86	3	J Clin Endocrinol Me	HCAPLUS
Di Matola, T	2000	85	4323	J Clin Endocrinol Me	HCAPLUS
Doval, H	1994	344	493	Lancet	MEDLINE
Farwell, A	1990	263	1526	JAMA	MEDLINE
Martino, E	1984	101	28	Ann Intern Med	MEDLINE
Martino, E	2001	2	240	Endocr Rev	
Martino, E	1991	14	847	J Endocrinol Invest	MEDLINE
Mulligan, D	1993	114	1114	Surgery	MEDLINE
Reiffel, J	1994	17	103	Clin Cardiol	MEDLINE

Robuschi, G	1986	9	287	J Endocrinol Invest	MEDLINE
Wang, Y	1987	65	679	J Clin Endocrinol Me	MEDLINE
Wu, S	1978	46	691	J Clin Endocrinol Me	HCAPLUS

L88 ANSWER 24 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:424213 HCAPLUS Full-text

DOCUMENT NUMBER: 135:179581

TITLE: Autoantibodies from patients with autoimmune thyroid disease do not interfere with the activity of the human iodide symporter gene stably transfected in CHO cells

AUTHOR(S): Tonacchera, Massimo; Agretti, Patrizia; Ceccarini, Giovanni; Lenza, Rosanna; Refetoff, Samuel; **Santini, Ferruccio; Pinchera, Aldo;** Chiovato, Luca; Vitti, Paolo

CORPORATE SOURCE: Dipartimento di Endocrinologia e Metabolismo, Ortopedia e Traumatologia, Medicina del Lavoro, Universita di Pisa, Pisa, 56124, Italy

SOURCE: European Journal of Endocrinology (2001), 144(6), 611-618

CODEN: EJOEEP; ISSN: 0804-4643

PUBLISHER: BioScientifica

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The human sodium iodide symporter (hNIS) is a candidate autoantigen in autoimmune thyroid diseases. To investigate the possible existence of autoantibodies able to interfere with the biol. activity of hNIS, an assay was developed using a cell line stably expressing hNIS. hNIS complementary cDNA cloned in pcDNA3 and a neomycin resistance gene vector were co-transfected into CHO cells. After selection with geneticin, a cell line termed PA4, showing the highest level of NaI25I uptake, was characterized. The time course of iodide uptake was evaluated by incubating PA4 cells with 10 µmol/l NaI and 0.1 µCi NaI25I for a period up to 90 min. The accumulation of iodide increased linearly between 2 and 10 min, reaching a plateau at 45 min. The curve of iodide efflux mirrored that of iodide influx. Both perchlorate and thiocyanate inhibited iodide uptake in PA4 cells in a dose-dependent manner starting from concns. as low as 0.01 and 0.1 µmol/l resp. and complete inhibition was obtained at concns. of 100 µmol/l perchlorate and 1000 µmol/l thiocyanate. The sensitivity of the inhibition assay was further improved using both inhibitors after 5 min incubation and in the absence of cold NaI. Included in the study were 42 patients with Graves' disease (25 had active hyperthyroidism, 10 were euthyroid, and 7 had hypothyroidism); 34 patients with Hashimoto's thyroiditis (1 was euthyroid, 4 had subclin. hypothyroidism, and 29 were overtly hypothyroid); and 19 with atrophic thyroiditis (all hypothyroid). Four out of 8 whole sera from patients with Hashimoto's thyroiditis, and 8 out of 25 whole sera from patients with Graves' disease caused an inhibition of iodide uptake in PA4 cells >20% but also in 4 out of 15 sera from normal subjects. This inhibition activity exerted by sera from patients and controls was lost after dialyzing against buffer. Accordingly, IgGs purified from sera of all patients with Graves' disease and with Hashimoto's thyroiditis or atrophic thyroiditis were devoid of any effect on iodide uptake. The authors thus believe that autoantibodies able to block the function of hNIS are very rare.

CC 15-8 (Immunochemistry)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
=====	=====	=====	=====	=====	=====
Ajjan, R	1998	83	1217	Journal of Clinical	HCAPLUS

Ajjan, R	2000	85	2020	Journal of Clinical	HCAPLUS
Chiovato, L	1990	71	40	Journal of Clinical	MEDLINE
Chiovato, L	1994	17	809	Journal of Endocrino	HCAPLUS
Dai, G	1996	379	458	Nature	HCAPLUS
Davies, T	2000		518	The Thyroid:a Fundam	
Endo, T	1996	228	199	Biochemical and Biop	HCAPLUS
Endo, T	1996	224	92	Biochemical and Biop	HCAPLUS
Ho, S	2000	85	3937	Journal of Clinical	
Konishi, J	1983	57	544	Journal of Clinical	MEDLINE
Kosugi, S	1996	227	94	Biochemical and Biop	HCAPLUS
Levy, O	1998	30	195	Journal of Bioenerge	HCAPLUS
Morris, J	1997	4	527	Thyroid	
Perret, J	1990	171	1044	Biochemical and Biop	HCAPLUS
Pohlenz, J	2000	85	2366	Journal of Clinical	HCAPLUS
Pohlenz, J	1998	101	1028	Journal of Clinical	HCAPLUS
Raspe, E	1995	132	399	European Journal of	HCAPLUS
Russo, D	1997	20	36	Journal of Endocrino	HCAPLUS
Saito, T	1997	82	3331	Journal of Clinical	HCAPLUS
Smanik, P	1996	226	339	Biochemical and Biop	HCAPLUS
Weiss, S	1984	114	1090	Endocrinology	HCAPLUS

L88 ANSWER 25 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:424209 HCAPLUS Full-text

DOCUMENT NUMBER: 135:147641

TITLE: Evidence for a role of the type III-iodothyronine
deiodinase in the regulation of 3,5,3'-
triiodothyronine content in the human central nervous
system

AUTHOR(S): *Santini, Ferruccio; Pinchera, Aldo*
; Ceccarini, Giovanni; Castagna, Maura; Rosellini,
Veronica; Mammoli, Claudia; Montanelli, Lucia; Zucchi,
Vanna; Chopra, Inder J.; Chiovato, Luca

CORPORATE SOURCE: Department of Endocrinology and Metabolism, University
of Pisa, Pisa, 56124, Italy

SOURCE: European Journal of Endocrinology (2001), 144(6),
577-583

CODEN: EJOEEP; ISSN: 0804-4643

PUBLISHER: BioScientifica

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Objective: Thyroid hormone is essential for maintaining normal neurol.
functions both during development and in adult life. Type III-iodothyronine
deiodinase (D3) degrades thyroid hormones by converting thyroxine and 3,5,3'-
triiodothyronine (T3) to inactive metabolites. A regional expression of D3
activity has been observed in the human central nervous system (CNS), and a
critical role for D3 has been suggested in the regulation of local T3 content
in concert with other enzymes. Design: This study was undertaken to further
characterize D3 activity in human CNS and to understand its role in the local
regulation of T3 content. Methods: Autoptic specimens from various areas of
human CNS were obtained 6-27 h postmortem from 14 donors who died from
cardiovascular accident, neoplastic disease or infectious disease. D3 was
determined by measuring the conversion of T3 to 3,3'-diiodothyronine. The T3
content was measured by RIA in ethanol exts., using a specific antiserum.
Results: High levels of D3 activity were observed in hippocampus and temporal
cortex, lower levels being found in the thalamus, hypothalamus, midbrain
cerebellum, parietal and frontal cortex, and brain stem. An inverse
relationship between D3 activity and T3 content in these areas was
demonstrated. Conclusion: We have concluded that D3 contributes to the local
regulation of T3 content in the human CNS.

CC 2-7 (Mammalian Hormones)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
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Arem, R	1993	42	1102	Metabolism	MEDLINE
Bates, J	1999	140	844	Endocrinology	HCAPLUS
Baumgartner, A	1997	16	25	Neuropsychopharmacol	HCAPLUS
Becker, K	1997	138	2989	Endocrinology	HCAPLUS
Bernal, J	1995	133	390	European Journal of	HCAPLUS
Calvo, R	1998	801	150	Brain Research	HCAPLUS
Campos-Barros, A	1996	81	2179	Journal of Clinical	HCAPLUS
Campos-Barros, A	1997	68	795	Journal of Neurochem	HCAPLUS
Chopra, I	1971	50	2033	Journal of Clinical	HCAPLUS
Chopra, I	1996		111	Werner and Ingbar's	
Escobar-Morreale, H	1995	96	2828	Journal of Clinical	HCAPLUS
Esfandiari, A	1992	131	1682	Endocrinology	HCAPLUS
Everts, M	1993	132	1278	Endocrinology	HCAPLUS
Huang, S	2000	343	185	New England Journal	MEDLINE
Huang, T	1986	35	272	Metabolism	HCAPLUS
Itagaki, Y	1990	71	340	Journal of Clinical	MEDLINE
Kaplan, M	1980	106	567	Endocrinology	HCAPLUS
Kaplan, M	1981	109	397	Endocrinology	HCAPLUS
Karmarkar, M	1993	57	291S	American Journal of	
Koehrle, J	1999	151	103	Molecular and Cellul	
Koopdonk-Kool, J	1996	81	2154	Journal of Clinical	HCAPLUS
Larsen, P	1981	2	87	Endocrine Reviews	HCAPLUS
Leonard, J	1996		125	Werner and Ingbar's	
Mori, K	1993	77	1198	Journal of Clinical	MEDLINE
Morreale de Escobar, G	1994	134	2410	Endocrinology	HCAPLUS
Obregon, M	1981	109	908	Endocrinology	HCAPLUS
Richard, K	1998	83	2868	Journal of Clinical	HCAPLUS
Rodriguez-Pena, A	1999	40	497	Journal of Neurobiol	HCAPLUS
Roti, E	1981	53	498	Journal of Clinical	HCAPLUS
Salvatore, D	1995	96	2421	Journal of Clinical	HCAPLUS
Santini, F	1992	130	2325	Endocrinology	HCAPLUS
Santini, F	1992	74	1366	Journal of Clinical	HCAPLUS
Santini, F	1999	84	493	Journal of Clinical	HCAPLUS
Silva, J	1984	73	898	Journal of Clinical	HCAPLUS
Stulp, M	1998	142	67	Molecular and Cellul	HCAPLUS
Tu, H	1999	140	784	Endocrinology	HCAPLUS
Visser, T	1983	71	992	Journal of Clinical	HCAPLUS
Visser, T	1986	9	17	Journal of Endocrino	
Wu, S	1976	43	682	Journal of Clinical	HCAPLUS

L88 ANSWER 26 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:706523 HCAPLUS Full-text

DOCUMENT NUMBER: 135:367023

TITLE: 3,3'-Diiodothyronine sulfate excretion in maternal urine reflects fetal thyroid function in sheep

AUTHOR(S): Wu, Sing-Yung; Huang, Wen-Sheng; Eisher, Delbert A.; Florsheim, Warner H.; Kashiwai, Kent; Polk, Daniel H.

CORPORATE SOURCE: Nuclear Medicine and Medicine Services, Department of Veterans' Affairs Medical Center, Log Beach, CA, 90822, USA

SOURCE: Pediatric Research (2001), 50(3), 358-364

CODEN: PEREBL; ISSN: 0031-3998

PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors have shown that there is significant fetal-to-maternal transfer of sulfated metabolites of thyroid hormone after fetal infusion of a pharmacol. amount T3 or sulfated T3 in late pregnancy in sheep. The transferred iodothyronine sulfoconjugate, i.e., 3,3'-diiodothyronine sulfate (T2S), of fetal origin appears in maternal sheep urine. The present study was carried out to assess the contribution of T2S of fetal origin to the urinary pool in ewes. Eighteen date-bred ewes (mean gestational age of 115 d) and their twin fetuses were divided into four groups. In group I (control), both ewes (M) and their fetuses (F) were sham operated for thyroidectomy (Tx). In group II, the ewes (MTx) and, in group III, the fetuses (FTx) were subjected to Tx. In group IV (MTx · FTx), both the ewe and fetus had Tx. After 10-12 d, fetal and/or maternal *hypothyroidism* were confirmed by serum thyroxine (<15 nmol/L) measurements. In addition, the authors infused radioactive T3 without disturbing the T3 pool in three singleton near-term fetuses and assessed the amount of radioactive iodothyronine that appeared in maternal urine (MU). After infusing [125I-3'],3,5-T3 via fetal vein to the near-term normal fetuses, radioactive T2S was identified as the major metabolite in MU by HPLC and T2S-specific antibody. MU T2S excretion (pmol/mmol creatinine) was significantly reduced by FTx and MTx-FTx but not by MTx. In addition, pos. correlations were found between MU T2S excretion and fetal serum thyroxine and T3 concns. but not with maternal serum thyroxine or T3 levels. T2S of fetal origin contributes significantly to the MU pool.

CC 2-7 (Mammalian Hormones)

IT Blood serum

Thyroid gland

Urine

(diiodothyronine sulfate excretion in maternal urine reflects fetal thyroid function in sheep)

IT 1041-01-6, Diiodothyronine 6893-02-3, Triiodothyronine
31135-55-4, Triiodothyronine sulfate

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(diiodothyronine sulfate excretion in maternal urine reflects fetal thyroid function in sheep)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
=====	=====	=====	=====	=====	=====
Bates, J	1999	140	844	Endocrinology	HCAPLUS
Castro, M	1985	76	1921	J Clin Invest	HCAPLUS
Chopra, I	1992	75	189	J Clin Endocrinol Me	MEDLINE
Chopra, I	1994		119	Thyroid Hormone Meta	HCAPLUS
Cortelazzi, D	1999	141	570	Eur J Endocrinol	HCAPLUS
Eelkman-Rooda, S	1988	9	125	J Immunoassay	MEDLINE
Galton, V	1999	103	979	J Clin Invest	HCAPLUS
Hurd, R	1993	133	1951	Endocrinology	HCAPLUS
Kaptein, E	1997	138	5136	Endocrinology	HCAPLUS
Kirk, R	1982		112	Experimental Design	
Lanni, A	1998	436	407	Eur J Physiol	HCAPLUS
Leonard, J	1997	7	147	Thyroid	MEDLINE
Maxwell, S	1990		242	Designing Experiment	
Mol, J	1985	117	1	Endocrinology	HCAPLUS
Moreno, M	1997	505	529	J Physiol	HCAPLUS
Nakamura, Y	1977	18	1112	J Nucl Med	HCAPLUS
Polk, D	1994	266	E892	Am J Physiol	HCAPLUS
Rudolph, A	1970	26	289	Circ Res	MEDLINE
Sack, J	1976	10	169	Pediatr Res	HCAPLUS
Santini, F	1993	133	105	Endocrinology	HCAPLUS
Santini, F	1999	84	493	J Clin Endocrinol Me	HCAPLUS

Visser, T	1994		85	Thyroid Hormone Meta	HCAPLUS
Wu, S	1998	178	886	Am J Obstet Gynecol	HCAPLUS
Wu, S	1993	265	E115	Am J Physiol	HCAPLUS
Wu, S	1995	268	E33	Am J Physiol	HCAPLUS
Wu, S	1999	277	E915	Am J Physiol	HCAPLUS
Wu, S	1992	131	1751	Endocrinology	HCAPLUS
Wu, S	1976	43	682	J Clin Endocrinol Me	HCAPLUS
Wu, S	1993	76	1625	J Clin Endocrinol Me	HCAPLUS
Wu, S	1994	78	1505	J Clin Endocrinol Me	HCAPLUS
Wu, S	2000	48	847	Pediatr Res	HCAPLUS
Wu, S	1992	2	101	Thyroid	MEDLINE
Wu, S	1991		293	Thyroid Hormone Meta	

L88 ANSWER 27 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1999:104153 HCAPLUS Full-text

DOCUMENT NUMBER: 130:262454

TITLE: Serum iodothyronines in the human fetus and the newborn: evidence for an important role of placenta in fetal thyroid hormone homeostasis

AUTHOR(S): *Santini, Ferruccio*; Chiovato, Luca; Ghirri, Paolo; Lapi, Paola; Mammoli, Claudia; Montanelli, Lucia; Scartabelli, Giovanna; Ceccarini, Giovanni; Coccoli, Luca; Chopra, Inder J.; Boldrini, Antonio; *Pinchera, Aldo*

CORPORATE SOURCE: Department of Endocrinology and Metabolism, University of Pisa, Pisa, 56124, Italy

SOURCE: Journal of Clinical Endocrinology and Metabolism (1999), 84(2), 493-498

CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The pattern of circulating iodothyronines in the fetus differs from that in the adult, being characterized by low levels of serum T3. In this study, concns. of various iodothyronines were measured in sera from neonates of various postconceptional age (PA). Obtained in cord sera at birth (PA, 24-40 wk), reflecting the fetal pattern, were compared with those found during extrauterine life in newborns of 5 days or more of postnatal life (PA, 27-46 wk). The main findings are: Starting at 30 wk of PA, serum levels increase linearly during extrauterine life; and at 40 wk, they are more than 200% of those measured in cord sera from newborns of equivalent PA. Serum reverse T3 (rT3) levels during fetal life are higher than those measured during extrauterine life; but they significantly decrease, starting at 30 wk of PA. Serum T3 sulfate (T3S) does not significantly differ between the two groups, showing the highest values at 28-30 wk of PA, and significantly decreasing at 30-40 wk. T3S levels are directly correlated with rT3, both in fetal and extrauterine life, whereas a significant neg. correlation between T3S and T3 is found only during extrauterine life. In conclusion: changes in serum concns. of iodothyronines in umbilical cord and during post-natal life indicate that maturation of extrathyroidal type I-iodothyronine monodeiodinase (MD) accelerates, starting at 30 wk of PA; high levels of type III-MD activity in fetal tissues prevent the rise of serum T3, whereas they maintain high levels of rT3 during intrauterine life; an important mechanism leading to the transition from the fetal to the postnatal thyroid hormone balance is a sudden decrease in type III-MD activity; because placenta contains a high amount of type III-MD, it is conceivable that placenta contributes to maintain low T3 and high rT3 serum concns. during fetal life and that its removal at birth is responsible for most changes in iodothyronine metabolism occurring afterwards.

CC 2-7 (Mammalian Hormones)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abuid, J	1973	52	1195	J Clin Invest	HCAPLUS
Anderson, R	1994		85	Thyroid hormone meta	
Burrow, G	1994	331	1072	N Engl J Med	HCAPLUS
Chiovato, L	1991	14	957	J Endocrinol Invest	MEDLINE
Chopra, I	1992	75	189	J Clin Endocrinol Me	MEDLINE
Chopra, I	1993	76	145	J Clin Endocrinol Me	HCAPLUS
Chopra, I	1974	54	583	J Clin Invest	HCAPLUS
Contempre, B	1993	77	1719	J Clin Endocrinol Me	HCAPLUS
Dubowitz, L	1997	77	1	J Pediatr	
Eelkman-Rooda, S	1988	124	740	Endocrinology	
Fisher, D	1977	33	59	Horm Res	
Fisher, D	1969	48	1670	J Clin Invest	HCAPLUS
Fisher, D	1986	9	1	Thyroid Today	
Fuse, Y	1996	8	1	Reprod Fertil Dev	HCAPLUS
Harris, A	1978	103	2216	Endocrinology	HCAPLUS
Huang, T	1988	23	196	Pediatr Res	HCAPLUS
Koopdonk-Kool, J	1996	81	2154	J Clin Endocrinol Me	HCAPLUS
Lopresti, J	1994	78	688	J Clin Endocrinol Me	HCAPLUS
Morreale De Escobar, G	1987	26	12	Horm Res	HCAPLUS
Mortimer, R	1996	81	2247	J Clin Endocrinol Me	HCAPLUS
Otten, M	1983	221	81	Science	HCAPLUS
Polk, D	1986	251	151	Am J Physiol	
Polk, D	1994	266	E892	Am J Physiol	HCAPLUS
Polk, D			223	Thyroid hormone meta	
Porterfield, S	1993	14	94	Endocr Rev	HCAPLUS
Richard, K	1997		S-117	Proc of the 70th Ann	
Roti, E	1981	53	498	J Clin Endocrinol Me	HCAPLUS
Sack, J	1976	10	169	Pediatr Res	HCAPLUS
Salvatore, D	1996	98	692	J Clin Invest	
Santini, F	1992	131	1689	Endocrinology	HCAPLUS
Santini, F	1992	131	2521	Endocrinology	HCAPLUS
Santini, F	1996	134	45	Eur J Endocrinol	HCAPLUS
Santini, F	1992	74	1366	J Clin Endocrinol Me	HCAPLUS
Santini, F	1993	76	1583	J Clin Endocrinol Me	MEDLINE
Thorpe-Beeston, J	1991	324	532	N Engl J Med	MEDLINE
van Wassenae, A	1993	129	139	Acta Endocrinol	HCAPLUS
Wu, S	1978	103	235	Endocrinology	HCAPLUS
Wu, S	1993	76	1625	J Clin Endocrinol Me	HCAPLUS
Wu, S	1986	8	43	J Dev Physiol	HCAPLUS

L88 ANSWER 28 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:58043 HCAPLUS Full-text

DOCUMENT NUMBER: 128:188475

TITLE: Acute effects of intravenous amiodarone on sulfate metabolites of thyroid hormones in arrhythmic patients

AUTHOR(S): Iervasi, Giorgio; Clerico, Aldo; Manfredi, Cristina; Sabatino, Laura; Biagini, Andrea; Chopra, Inder J.

CORPORATE SOURCE: Laboratory of Cardiovascular Endocrinology, CNR Institute of Clinical Physiology, Pisa, 56100, Italy

SOURCE: Clinical Endocrinology (Oxford) (1997), 47(6), 699-705
CODEN: CLECAP; ISSN: 0300-0664

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Factors that contribute to the remarkably rapid decrease in serum T3 and increase in reverse T3 (rT3) levels during illness, fasting, or treatment with some drugs (e.g., amiodarone) are not clear. To understand better the effect

of acute amiodarone administration on T3 metabolism, especially the sulfation pathway, the authors performed a prospective study in 8 arrhythmic in-patients treated with a loading dose of amiodarone. Amiodarone was administered by i.v. infusion of 20 mg/kg/day on day 1 and 10 mg/kg/day on day 2, followed by 600 mg/day orally throughout the study. Two serum samples for amiodarone and hormone assays (thyroid hormones, TSH, and the sulfate metabolites of 3'-T1, 3,3'-T2, and T3) were collected before the start of therapy, every 12 h during the first 3 days of amiodarone administration, and then once a day for 2-10 days. Eight patients (4 men and 4 women, aged 44-82 yr), who were treated with amiodarone because of cardiac dysrhythmia, were enrolled in the study. Serum concns. of total T4 significantly increased in the last 3 days of the study (ANOVA). However, serum total T3 progressively and significantly decreased throughout the study (ANOVA). Serum free thyroid hormone concns. (freeT3 and freeT4) did not significantly change during the study. Serum rT3 (ANOVA) and TSH (ANOVA) rapidly and progressively increased throughout the study. Starting from the first 24 h, serum concns. of T3 sulfate (T3-S) significantly and progressively increased from (mean) 0.057 nM under basal conditions to 0.089 nM after 5 days of amiodarone therapy (ANOVA). Since total T3 levels progressively decreased throughout the study, the ratio of the T3-S and total T3 values progressively increased from 4.8% under basal conditions to 10.6% after 5 days of amiodarone therapy (ANOVA, repeated measures). Basal serum concns. of sulfate metabolites of T2 (T2-S, 2.22 nM) and T1 (T1-S, 1.29 nM) did not significantly change throughout the study. The authors' data indicate that a loading dose of i.v. amiodarone in patients with cardiac dysrhythmias is followed by a very rapid and progressive increase in circulating T3-S levels, possibly due to an inhibition of type 1-iodothyronine de-iodinase. Since T2-S and T1-S, common final metabolites of the thyroid hormone sulfation pathways remained unchanged, the authors' data suggest that the total amount of thyroid hormone degraded by sulfation pathways remains unaltered during amiodarone treatment. Finally the authors' findings are compatible with the view that sulfation represents an important pathway for T3 metabolism in vivo in man.

CC 1-8 (*Pharmacology*)

Section cross-reference(s): 2

IT Heart, *disease*

(arrhythmia; acute effects of i.v. amiodarone on sulfate metabolites of thyroid hormones in arrhythmic patients)

IT 5817-39-0, Reverse triiodothyronine 6893-02-3, Triiodothyronine
9002-71-5, Thyrotropin 31135-55-4, Triiodothyronine
sulfate 64192-57-0 64192-58-1

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(acute effects of i.v. amiodarone on sulfate metabolites of thyroid hormones in arrhythmic patients)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Chopra, I	1992	75	189	Journal Clinical End	MEDLINE
Chopra, I	1996			Program Annual Meeti	
de Groot, L	1996		61	The thyroid and its	
Eedkman, R	1988	124	740	Endocrinology	
Eedkman, R	1984	9	125	Journal of Immunoass	
Engler, D	1984	5	151	Endocrine Revue	HCAPLUS
Figge, H	1990	30	588	Journal of Clinical	MEDLINE
Gill, J	1992	43	69	Drugs	MEDLINE
Hershman, J	1986	111	193	Acta Endocrinologica	MEDLINE
Iervasi, G	1996	26	382	European Journal of	HCAPLUS
Iervasi, G	1997	82	275	Journal Clinical End	HCAPLUS
Kannan, R	1990	122	249	Acta Endocrinologica	HCAPLUS

Lambert, M	1982	55	1058	Journal Clinical End	MEDLINE
Leonard, J	1995		125	Werner and Ingbar's	
Lopresti, J	1991	73	703	Journal Clinical End	MEDLINE
Lopresti, J	1994	78	688	Journal Clinical End	HCAPLUS
Lopresti, J	1987			Significance of trii	
Manfredi, C	1995	15	87	International Journa	HCAPLUS
Mol, J	1985	117	1	Endocrinology	HCAPLUS
Mol, J	1985	117	8	Endocrinology	HCAPLUS
Otten, M	1983	221	81	Science	HCAPLUS
Polikar, R	1993	87	1435	Circulation	MEDLINE
Unger, J	1993	233	435	Journal of Internal	MEDLINE
Visser, T	1983	112	1547	Endocrinology	HCAPLUS

L88 ANSWER 29 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1996:519721 HCAPLUS Full-text

DOCUMENT NUMBER: 125:191543

TITLE: Transfection with the cDNA of the human thyrotropin receptor of a poorly differentiated rat thyroid cell line (FRT)

AUTHOR(S): Elisei, R.; Pinchera, A.; Chiovato, L.; Mammoli, C.; Agretti, P.; Romei, C.; Santini, F.; Bendinelli, G.; Fiore, E.; et al.

CORPORATE SOURCE: Istituto di Endocrinologia, University Pisa, 56018, Italy

SOURCE: Journal of Endocrinological Investigation (1996), 19(4), 230-235

CODEN: JEIND7; ISSN: 0391-4097

PUBLISHER: Editrice Kurtis s.r.l.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A cell line derived from the Fisher rat thyroid (FRT), that does not have functional TSH receptor, was stably transfected with the cDNA of the human TSH receptor (hTSH-R). In wild FRT cells TSH (1-1000 mU/l) was unable to increase cAMP production, while 10-10000 nmol/l forskolin elicited a 10-30 fold cAMP stimulation. Two of the transfected clones were responsive to TSH in terms of cAMP production. In particular, the FRT-R3 transfected clone showed the highest sensitivity to the hormone with a 10 fold cAMP increase over the basal at 100 mU/l TSH. The Northern blot anal. using a 2.4 kbp cDNA probe for the hTSH-R showed a band corresponding to the mRNA of TSH receptor in FRT-R3 cells, but not in wild FRT cells. In both cell types TSH was ineffective in stimulating growth assayed by 3H-thymidine incorporation into DNA. Hybridization with a probe for thyroperoxidase on polymerase chain reaction products after reverse transcription of mRNA showed that FRT-R3, as well as FRT cells, do not have a transcript for thyroperoxidase. In conclusion, the insertion of the hTSH-R cDNA in the genome of poorly differentiated rat thyroid cells results in the recovery of TSH-dependent adenylate cyclase, but not other differentiated thyroid cell functions.

CC 13-2 (Mammalian Biochemistry)

Section cross-reference(s): 1

L88 ANSWER 30 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1994:315493 HCAPLUS Full-text

DOCUMENT NUMBER: 120:315493

TITLE: Studies on the in vitro cytotoxic effect of amiodarone

AUTHOR(S): Chiovato, Luca; Martino, Enio; Tonacchera, Massimo; Santini, Ferruccio; Lapi, Paola; Mammoli, Claudia; Braverman, Lewis E.; Pinchera, Aldo

CORPORATE SOURCE: Ist. Endocrinol., Univ. Pisa, Tirrenia-Pisa, 56018, Italy

SOURCE: Endocrinology (1994), 134(5), 2277-82

DOCUMENT TYPE: Journal
LANGUAGE: English

AB Amiodarone, a potent antiarrhythmic drug, contains 37.2% iodine by weight and may induce either hypo- or hyperthyroidism. The high iodine content of amiodarone may be responsible for both complications, but a cytotoxic effect of the drug on the thyroid resulting in thyroiditis has been reported. In the present study the cytotoxic effect of amiodarone was evaluated in three culture systems with different biol. properties: 1) a strain of rat thyroid cells (FRTL-5 cells) that maintains most differentiated functions of normal thyroid cells, including an active iodide pump, but an inability to organify iodide; 2) a line of Chinese hamster ovary (CHO) fibroblasts; and 3) freshly prepared primary cultures of human thyroid follicles (hTF)h that trap and organify iodide. Cells were radiolabeled with ^{51}Cr and incubated for 24 h with medium alone, medium plus amiodarone (3.75-200 μM , medium plus an iodinated radiog. contrast agent (sodium diatrizoate; 7.5-200 μM), or medium plus potassium iodide (7.5-300 μM). At concns. ranging from 75-200 μM , amiodarone induced a significant and dose-dependent release of ^{51}Cr in FRTL-5 cells. In contrast, diatrizoate or KI had no cytotoxic effect on FRTL-5 cells. In the same molar concns., amiodarone was also cytotoxic to CHO cells. In hTF, the release of ^{51}Cr produced by amiodarone occurred at a lower concentration (37.5 vs. 75 μM) and was significantly greater than that in FRTL-5 cells. The cytotoxic effects of amiodarone in hTF was partially, but significantly, reduced by methimazole, an inhibitor of iodide organification. In the FRTL-5 cell culture system, amiodarone also produced a dramatic inhibition of TSH-stimulated cell growth. This growth-inhibiting effect of amiodarone was evident at low concns. (3.75-7.5 $\mu\text{mol/L}$) of the drug, which did not produce cytotoxicity. In conclusion, 1) amiodarone had a cytotoxic effect in CHO fibroblasts, a nonthyroid cell line; 2) this cytotoxic effect occurred in thyroid cells independent of their ability to organify iodide; 3) however, the toxic effect of amiodarone was greater and occurred at a low molar concentration in freshly prepared human thyroid follicles that trap and organify iodide; and 4) in the latter culture system, methimazole, an inhibitor of iodide organification, partially, but significantly, reduced the cytotoxic effect of amiodarone. These data suggest that thyroid cytotoxicity produced by amiodarone is mainly due to a direct effect of the drug on thyroid cells, but excess iodide released from the drug may contribute to its toxic action.

CC 1-8 (Pharmacology)

L88 ANSWER 31 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1994:646873 HCAPLUS Full-text

DOCUMENT NUMBER: 121:246873

TITLE: Uptake of triiodothyronine sulfate and suppression of thyrotropin secretion in cultured anterior pituitary cells

AUTHOR(S): Everts, M. E.; Visser, T. J.; van Buuren, J. C. J.; Docter, R.; de Jong, M.; Krenning, E. P.; Hennemann, G.

CORPORATE SOURCE: Med. Sch., Erasmus Univ., Rotterdam, 3000 DR, Neth.
SOURCE: Metabolism, Clinical and Experimental (1994), 43(10), 1282-6

CODEN: METAAJ; ISSN: 0026-0495

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To investigate the uptake of triiodothyronine sulfate (T3S) and its effect on TSH-releasing hormone (TRH)-induced TSH secretion, anterior pituitary cells were isolated from euthyroid rats and cultured for 3 days in medium containing 10% fetal calf serum. Incubation was performed at 37° in medium containing

0.5% bovine serum albumin (BSA). Exposure of the pituitary cells to TRH (0.1 μ mol/L) for 2 h stimulated TSH secretion by 176%. This effect was reduced by approx. 45% after a 2-h preincubation with T3 (0.001 to 1 μ mol/L). A significant inhibitory effect of T3S on TRH-induced TSH release was only observed at a concentration of 1 μ mol/L. The uptake of [125I]T3 after 1 h of incubation was reduced by 40% by simultaneous addition of 10 nmol/L unlabeled T3, whereas 1 μ mol/L T3S was required to obtain a reduction of the [125I]T3 uptake by 34%. The amount of T3 present in the unlabeled T3S preparation was 0.25% as determined by RIA. When pituitary cells were incubated for 1 h with [125I]T3S or [125I]T3 (both 50,000 cpm/0.25 mL), the uptake of [125I]T3S expressed as a percentage of the dose was 0.04%, whereas that of [125I]T3 amounted to 3.0%. In contrast, when hepatocytes were incubated for 1 h with [125I]T3S, the uptake amounted to 5.1%, whereas that of [125I]T3 was 22.1%. Furthermore, [125I]T3S was a rapidly deiodinated (iodide production, 14.9%) as [125I]T3 (12.1%) by hepatocytes. It is concluded that T3S is poorly taken up by pituitary cells, and the suppressive effect of high concns. of T3S on TRH-induced TSH secretion and on [125I]T3 uptake can be explained by slight contamination with T3. Thus, it appears that T3S has only a minor biol. effect, if any, on the pituitary.

CC 2-7 (Mammalian Hormones)

IT 31135-55-4, Triiodothyronine sulfate

RL: BAC (Biological activity or effector, except adverse); BSU

(Biological study, unclassified); BIOL (Biological study)

(triiodothyronine sulfate uptake and suppression of TSH secretion in cultured anterior pituitary cells)

L88 ANSWER 32 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1995:341437 HCAPLUS Full-text

DOCUMENT NUMBER: 122:130416

TITLE: Detection of antibodies blocking thyrotropin effect using Chinese hamster ovary cells transfected with the cloned human TSH receptor

AUTHOR(S): Chiovato, L.; Vitti, P.; Bendinelli, G.; Santini, F.; Fiore, E.; Capaccioli, A.; Tonacchera, M.; Mammoli, C.; Ludgate, M.; Pinchera, A.

CORPORATE SOURCE: Istituto di Endocrinologia, University of Pisa, Pisa, Italy

SOURCE: Journal of Endocrinological Investigation (1994), 17(10), 809-16

CODEN: JEIND7; ISSN: 0391-4097

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Chinese hamster ovary (CHO) cells transfected with the cloned human TSH receptor (CHO-R) were used to develop an assay to detect thyroid autoantibodies blocking the TSH-dependent cAMP production (TSHBAb). The study group included patients with goitrous Hashimoto's thyroiditis (HT) and subjects with atrophic thyroiditis (AT). In the HT group, both patients with subclin. hypothyroidism (HT-SH) and overt hypothyroidism (HT-H) were represented. Normal subjects served as controls. IgG was prepared from serum by double chromatog. on DEAE-Sephadex. CHO-R cells were seeded in 96-well plates and were cultured for 48 h before the assay in RPMI-1640 medium plus 1 mmol/L glutamine, 10% fetal calf serum, and 0.4 g/L geneticin. In the assay for TSHBAb, CHO-R cells were incubated with IgG alone (0.5-2 mg/mL), TSH alone (0.2-625 mU/L), or IgG plus TSH; all samples were diluted in hypotonic medium containing 0.5 mmol/L isobutyl-methylxanthine (IBMX). After 2 h of incubation at 37° in 5% CO₂ -95% air atmospheric, TSH-stimulation was quantified by measuring extracellular cAMP by a RIA. IgGs from normal subjects did not modify the stimulation of adenylate cyclase produced by TSH, the results obtained ranging between -30% and +18% (mean -3%). All IgGs producing an

inhibition >2SD from the mean of controls (>25%) were considered pos. for blocking antibodies. TSHBAb were detected in 1/8 (12.5%) patients with HT-SH, in 7/30 (23.3%) with HT-H, and in 16/47 (34.0%) patients with AT. When the same IgGs were tested in FRTL-5 cells, TSHBAb were detected in 1/8 (12.5%) patients with HT-SH, in 5/30 (16.6%) with HT-H, and in 15/47 (31.9%) with AT. TSHBAb results in CHO-R cells showed a good correlation with those in FRTL-5 cells, but 3/24 IgGs were pos. for TSHBAb in CHO-R cells and neg. in FRTL-5 cells. Using the radioreceptor assay, TSH-binding inhibiting antibodies were detected in 17/24 (70.8%) sera that contained TSHBAb when tested in the CHO-R cell system. Thyroid stimulating antibody (TSAb) and TSHBAb, that coexisted in 5 IgGs, were simultaneously detected using CHO-R cells. These IgGs belonged to patients in whom spontaneous hypothyroidism developed after hyperthyroidism, or vice versa. Thus, a new in vitro assay for the detection of TSHBAb was developed using CHO-R cells. The sensitivity of this assay is slightly greater than that obtained in FRTL-5 cells and definitely greater than that of the radioreceptor assay. CHO-R cells have the advantage of expressing the human TSH receptor and of requiring less cumbersome procedures for cell culture than FRTL-5 cells.

CC 15-1 (Immunochemistry)

L88 ANSWER 33 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1994:672223 HCAPLUS Full-text

DOCUMENT NUMBER: 121:272223

TITLE: Bioactivity of sulfoconjugated iodothyronines

AUTHOR(S): Spaulding, Stephen Waasa

CORPORATE SOURCE: College Med., SUNY, Buffalo, NY, 14215, USA

SOURCE: Thyroid Horm. Metab. [Proc. Int. Conf.], 2nd (1994), Meeting Date 1993, 139-53. Editor(s): Wu, Sing-Yung; Visser, Theo J. CRC: Boca Raton, Fla.

CODEN: 60ROAZ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review, with 22 refs., on evidence that deconjugation of triiodothyronine sulfate occurs in vivo and in vitro and on some of the ways used to test whether triiodothyronine sulfate has a direct action in in-vitro systems where thyroid hormones are known to have an effect.

CC 2-0 (Mammalian Hormones)

IT 31135-55-4, Triiodothyronine sulfate

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study) (bioactivity of sulfoconjugated iodothyronines)

L88 ANSWER 34 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1994:672222 HCAPLUS Full-text

DOCUMENT NUMBER: 121:272222

TITLE: The role of sulfation and desulfation in thyroid hormone metabolism

AUTHOR(S): Chopra, Inder J.; Santini, Ferruccio; Wu, Sing-Yung; Hurd, Robert E.

CORPORATE SOURCE: Sch. Med., Univ. California, Los Angeles, CA, 90024, USA

SOURCE: Thyroid Horm. Metab. [Proc. Int. Conf.], 2nd (1994), Meeting Date 1993, 119-38. Editor(s): Wu, Sing-Yung; Visser, Theo J. CRC: Boca Raton, Fla.

CODEN: 60ROAZ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review, with 35 refs., on RIAs for triiodothyronine sulfate (T3S), thyroxine sulfate and reverse triiodothyronine sulfate, the interaction between sulfoconjugates of iodothyronines and iodothyronine monodeiodinases, T3S in

hypothyroidism, T3S desulfation in tissues, triiodothyronine sulfation in tissues, and the biol. activity of T3S.

CC 2-0 (Mammalian Hormones)

IT 31135-55-4, *Triiodothyronine sulfate*

77074-49-8, Thyroxine sulfate 79349-15-8

RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)

(sulfation and desulfation in thyroid hormone metabolism)

L88 ANSWER 35 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1994:1066 HCAPLUS Full-text

DOCUMENT NUMBER: 120:1066

TITLE: A study of the 3,5,3'-triiodothyronine sulfation activity in the adult and the fetal rat

AUTHOR(S): Hurd, Robert E.; Santini, Ferruccio; Lee, Brenda; Naim, Pauline; Chopra, Inder J.

CORPORATE SOURCE: Cent. Health Sci., Univ. California, Los Angeles, CA, 90024-1682, USA

SOURCE: Endocrinology (1993), 133(5), 1951-5
CODEN: ENDOAO; ISSN: 0013-7227

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors have employed an in vitro assay for studying the T3 sulfation activity in rat tissues. The assay measures by RIA the generation of T3 sulfate (T3S) during incubation of T3 with cytosol of rat tissues as the source of phenol sulfotransferase(s) and 3-phosphoadenosine-5'-phosphosulfate as the sulfate donor. The conversion of T3 to T3S proceeded rapidly for 30 min at 37°, and the optimal pH of the reaction was 8.0. Heating the cytosol at 44° for 15 min decreased T3S production to 63% of its value at 37°. T3 sulfation activity was plentiful in rat liver, brain, and kidney, but little activity was demonstrable in other tissues. The Km and maximum velocity of the hepatic conversion of T3 to T3S were 114 µM and 159 pmol/mg protein/h, resp. There was a marked inhibition of the conversion of T3 to T3S with salicylamide, 3'-monoiodothyronine, thyronine, and rT3; the IC50 of these inhibitors was: approximated 15, < 0.1, 9.5, and 43 µM, resp. On day 17 of gestation, the T3 to T3S conversion activity was more abundant in fetal skin than in other fetal tissues. However, the activity decreased in fetal skin while it increased in fetal liver, kidney, and brain nearer to term on day 20. Placenta demonstrated lower T3 to T3S conversion activity than did several fetal or maternal tissues. There was no effect of *hypothyroidism* or hyperthyroidism on T3 sulfation activity. Apparently, T3 sulfation activity in the rat is: (1) most abundant in liver, kidney, and brain tissues of the adult; (2) inhibited more avidly by 3'-monoiodothyronine than by other thyronines; (3) very abundant in fetal skin early in gestation; and (4) little affected by the thyroidal status of the animal.

CC 2-7 (Mammalian Hormones)

IT 31135-55-4

RL: ANT (Analyte); ANST (Analytical study)
(determination of, by RIA)

L88 ANSWER 36 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1991:574095 HCAPLUS Full-text

DOCUMENT NUMBER: 115:174095

TITLE: Selective inhibition of glucuronidation by 2,2,2-triphenylethyl-UDP in isolated rat hepatocytes: conjugation of harmol, 3,3',5-triiodothyronine, and N-hydroxy-2-acetylaminofluorene

AUTHOR(S): Noort, Daan; Meijer, Ellen A.; Visser, Theo J.; Meerman, John H. N.; Van der Marel, Gijs A.; Van Boom,

Jacques H.; Mulder, Gerard J.
CORPORATE SOURCE: Cent. Bio-Pharm. Sci., Univ. Leiden, Leiden, 2300 RA, Neth.
SOURCE: Molecular Pharmacology (1991), 40(2), 316-20
CODEN: MOPMA3; ISSN: 0026-895X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 2,2,2-Triphenylethyl-UDP (TPEU) was synthesized as an analog of the transition state of the glucuronidation reaction catalyzed by UDP-glucuronosyltransferase; it contains both a uridine and an acceptor substrate moiety. It inhibits rat liver microsomal UDP-glucuronosyltransferase [Eur. J. Biochem. 188:309-312 (1990)]. In the present work, TPEU was tested as an inhibitor of glucuronidation in intact rat hepatocytes. Two phenols (harmol and 3,3',5-triiodothyronine) and a hydroxamic acid (N-hydroxy-2-acetylaminofluorene) were used as substrates for glucuronidation. The glucuronidation of these substrates was strongly decreased by TPEU at 0.3-5 mM. Up to 5 mM TPEU did not kill the cells, as shown by unimpaired trypan blue exclusion at the end of the incubation. When glucuronidation was inhibited, the sulfation of harmol increased, as did the production of reactive species generated from N-hydroxy-2-acetylaminofluorene that bind to cellular macromols. This indicates that a decreased substrate consumption by loss of glucuronidation leads to increased conversion by competing pathways. The results show, therefore, that TPEU is an effective inhibitor of glucuronidation in this cellular system in vitro.
CC 1-4 (Pharmacology)
Section cross-reference(s): 4
IT 27067-62-5, Harmol sulfate 31135-55-4
RL: FORM (Formation, nonpreparative)
(formation of, in hepatocytes, triphenylethyl-UDP inhibition of glucuronidation in relation to)

L88 ANSWER 37 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1991:527851 HCAPLUS Full-text
DOCUMENT NUMBER: 115:127851
TITLE: Regulation by TSH and thyroid stimulating antibodies of TPO expression
AUTHOR(S): Chiovato, L.; Vitti, P.; Mammoli, C.; Santini, F.; Tonacchera, M.; Lapi, P.; Cucchi, P.; Carayon, P.; Pinchera, A.
CORPORATE SOURCE: Univ. Pisa, Pisa, 56018, Italy
SOURCE: Colloque INSERM (1990), 207(Thyroperoxidase Thyroid Autoimmun.), 43-51
CODEN: CINMDE; ISSN: 0768-3154
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mechanisms responsible for the expression of the thyroid microsomal/peroxidase autoantigen (M/TPO-Ag) were studied in FRTL-5 cells and in primary cultures of human thyroid cells prepared from Graves' or nontoxic goiters. The expression of M/TPO-Ag in thyroid cells is dependent on TSH stimulation, through pathways which involve cAMP production, mRNA formation and protein synthesis. Thyroid-stimulating antibody reproduces this effect of TSH, and estradiol and NaI have no direct influence on the expression of the M/TPO-Ag.
CC 2-7 (Mammalian Hormones)
Section cross-reference(s): 15

L88 ANSWER 38 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1989:437589 HCAPLUS Full-text
DOCUMENT NUMBER: 111:37589
TITLE: Antibodies to human thyroid peroxidase in autoimmune

thyroid disease: studies with a cloned recombinant complementary deoxyribonucleic acid epitope
AUTHOR(S): Ludgate, M.; Mariotti, S.; Libert, F.; Dinsart, C.; Piccolo, P.; Santini, F.; Ruf, J.; Pinchera, A.; Vassart, G.

CORPORATE SOURCE: Univ. Libre Bruxelles, IRIBHN, Brussels, Belg.
SOURCE: Journal of Clinical Endocrinology and Metabolism (1989), 68(6), 1091-6
CODEN: JCEMAZ; ISSN: 0021-972X

DOCUMENT TYPE: Journal
LANGUAGE: English

AB Previous studies carried out by screening a λ gt11 human thyroid cDNA library with serum samples from selected patients with Hashimoto's thyroiditis and a polyclonal antibody to porcine thyroid peroxidase (TPO) confirmed, at the mol. level, that TPO is a major component of the thyroid microsomal antigen (M). That investigation led to the isolation of a clone (C2) which encodes an 85-amino acid segment of TPO and harbors a major epitope recognized by serum from several patients with autoimmune thyroid disease that contained anti-M autoantibodies (MAb). In this study, C2 antigen that was produced as a β -galactosidase fusion protein was used to establish an enzyme-linked immunoabsorbent assay for the detection of anti-C2 autoantibodies (C2Ab). C2Ab then were assayed in 191 patients with different autoimmune and nonautoimmune thyroid disorders, and 50 patients with nonthyroidal autoimmune diseases. The results were compared with the titers of anti-TPO antibodies (TPOAb; as detected by monoclonal antibody-assisted RIA) and MAb (as detected by passive hemagglutination). Pos. C2Ab was found in the serum of 85 of 136 (63%) patients whose serum contained TPOAb and/or MAb. A pos. correlation was found between the levels of C2Ab and those of TPOAb or MAb, which was independent of the type of underlying autoimmune thyroid disorder. Low levels of C2Ab also were found in 10 of 105 (9%) serum samples that did not contain TPOAb. Western blot anal. carried out on the latter samples showed that in 2 samples the apparent C2Ab reactivity was due to the presence of antibodies reacting with β -galactosidase. Thus, the authors confirmed the validity of screening λ gt11 cDNA human thyroid libraries to better characterize thyroid autoantigens and demonstrated the feasibility of using recombinant proteins to establish diagnostic assays for autoantibodies.

CC 15-3 (Immunochemistry)

L88 ANSWER 39 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:405445 HCAPLUS Full-text

DOCUMENT NUMBER: 111:5445

TITLE: Thyroid autoantigens and their relevance in the pathogenesis of thyroid autoimmunity

AUTHOR(S): Pinchera, Aldo; Mariotti, Stefano; Vitti, Paolo; Marcocci, Claudio; Chiovato, Luca; Fenzi, Gianfranco; Santini, Ferruccio

CORPORATE SOURCE: Univ. Pisa, Tirrenia, 56018, Italy

SOURCE: Biochimie (1989), 71(2), 237-45

CODEN: BICMBE; ISSN: 0300-9084

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 45 refs. of the TSH receptor, thyroglobulin, and thyroid peroxidase as autoantigens important in the pathogenesis of thyroid autoimmunity. Thyroid autoantibodies may interfere in some peculiar thyroid function activities.

CC 15-0 (Immunochemistry)

L88 ANSWER 40 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:405279 HCAPLUS Full-text

DOCUMENT NUMBER: 111:5279
TITLE: Effects of thyroid hormone on sulfate activation pathway in neonatal *hypothyroid* rats
AUTHOR(S): Iwamura, Chiyo
CORPORATE SOURCE: Med. Sch., Osaka City Univ., Osaka, Japan
SOURCE: Osaka-shi Igakkai Zasshi (1988), 37(2), 367-83
CODEN: OIGZDE; ISSN: 0386-4103
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

- AB In neonatal *hypothyroid* rats, there is a reduction of sulfuric compds., sulfatide or acid mucopolysaccharide. Sulfuric compds. are sulfated by PAPS (3'-phosphoadenosine 5'-phosphosulfate), synthesized by the sulfate activation pathway. The effect of thyroid hormone on the sulfate activation pathway in neonatal *hypothyroid* rats was studied. Incorporation of $^{35}\text{S}\text{O}_4$ into brain lipid in the *hypothyroid* rats was decreased by 65% of the control rats and the cartilage level of the isotope was decreased by 64%. The in vitro incorporation of $^{35}\text{S}\text{O}_4$ into 5'-phosphoadenosine sulfate (APS), PAPS and endogeneous acceptor was determined. The accumulated ^{35}S PAPS in exts. of the neonatal *hypothyroid* rats was decreased. The accumulated ^{35}S APS was increased and the percentage of conversion of APS to PAPS was decreased in the neonatal *hypothyroid* rats. ATP sulfurylase assay in the cartilage or brain exts. showed no differences. Neonatal *hypothyroidism* reduced the concentration of sulfatide in brain lipid. In the cartilage of *hypothyroid* rats, the concentration of mucopolysaccharides with low sulfate content was increased. ATP sulfurylase activity in cultured chondrocytes with T3, T4-free medium showed no difference from those with T3-supplemented medium, whereas the APS kinase activity in cultured chondrocytes with T3, T4-free medium was reduced. Apparently, in cases of neonatal *hypothyroidism*, the reduction of sulfatide or mucopolysaccharide results from a decreased PAPS synthesis and a defective conversion of APS to PAPS. Thyroid hormones might affect APS kinase and alter the metabolism of sulfuric compds.
- CC 14-8 (Mammalian Pathological Biochemistry)
- ST neonate *hypothyroidism* sulfate activation thyroid hormone
- IT Brain, composition
(lipids of, sulfate incorporation into, in neonatal *hypothyroidism*)
- IT Sulfatides
RL: BIOL (Biological study)
(of brain, in neonatal *hypothyroidism*)
- IT Sulfolipids
RL: BIOL (Biological study)
(of brain, sulfate incorporation into, in neonatal *hypothyroidism*)
- IT Chondrocyte
(phosphoadenosine sulfate kinase of, in neonatal *hypothyroidism*, thyroid hormones in relation to)
- IT Newborn
(sulfate activation pathway in *hypothyroidism* in, thyroid hormones effects on)
- IT Thyroid hormones
RL: BIOL (Biological study)
(sulfate activation pathway in neonatal *hypothyroidism* response to)
- IT *Hypothyroidism*
(sulfate activation pathway in neonatal, thyroid hormones effects on)
- IT Cartilage
(sulfate incorporation into, in neonatal *hypothyroidism*)
- IT Mucopolysaccharides, biological studies
RL: BIOL (Biological study)
(acid, in neonatal *hypothyroidism*)

IT 14808-79-8, Sulfate, biological studies
 RL: BIOL (Biological study)
 (activation pathway, in neonatal *hypothyroidism*, thyroid hormone effects on)

IT 482-67-7, 3'-Phosphoadenosine 5'-phosphosulfate
 RL: FORM (Formation, nonpreparative)
 (formation of, defect in, in neonatal *hypothyroidism*)

IT 9012-39-9, ATP sulfurylase
 RL: BIOL (Biological study)
 (of brain and cartilage, in neonatal *hypothyroidism*)

IT 9012-38-8, 5'-Phosphoadenosine sulfate kinase
 RL: BIOL (Biological study)
 (of chondrocytes, in neonatal *hypothyroidism*, thyroid hormones in relation to)

IT 51-48-9, Thyroxine, biological studies 6893-02-3, *Triiodothyronine*
 RL: BIOL (Biological study)
 (sulfate activation pathway in neonatal *hypothyroidism* response to)

IT 485-84-7
 RL: BIOL (Biological study)
 (sulfate incorporation into, in neonatal *hypothyroidism*)

L88 ANSWER 41 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1981:564973 HCAPLUS Full-text

DOCUMENT NUMBER: 95:164973

TITLE: Urinary excretion of free and conjugated 3',5'-diiodothyronine and 3,3'-diiodothyronine

AUTHOR(S): Faber, J.; Busch-Sorensen, M.; Rogowski, P.; Kirkegaard, C.; Siersbaek-Nielsen, K.; Friis, T.

CORPORATE SOURCE: Med. Dep. E, Frederiksberg Hosp., Copenhagen, DK-2000, Den.

SOURCE: Journal of Clinical Endocrinology and Metabolism (1981), 53(3), 587-93

CODEN: JCEMAZ; ISSN: 0021-972X

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Radioimmunoassays (RIAs) for estimation of 3',5'-diiodothyronine (I) and 3,3'-diiodothyronine (II) in human urine were established. The urinary excretion of both glucuronide and sulfate conjugates of I, II and of T4, T3, and rT3 were estimated by means of enzymic deconjugation. In healthy controls, the mean excretion (picomoles/24 h) of free T4 was 1820, free T3 813, free rT3 77, free I 13, and free II 674. The total excretion of free and conjugated T4 was 2941, T3 1283, rT3 791, I 709, and II 2688. Significant amts. of sulfated T4 and T3 could not be demonstrated, whereas the excretion of sulfated rT3 was higher than that of glucuronidated rT3. In contrast, glucuronidated and sulfated I as well as glucuronidated and sulfated II were found in the urine in equal amts. In hyperthyroidism, the excretions of free and glucuronidated iodothyronines were increased, whereas the increase of the excretions of sulfated iodothyronines were less pronounced, only reaching statistical significance for II. In *hypothyroidism*, the excretions of both free, glucuronidated, and sulfated iodothyronines were reduced. Significant amts. of sulfated T4 and T3 could not be demonstrated in urine from hyperthyroid or *hypothyroid* patients. The amts. of free iodothyronines excreted in the urine vary considerably, suggesting active renal handling. The amts. of urinary glucuronidated and sulfated conjugates of the different iodothyronines studied vary considerably and are affected by thyroid function.

CC 9-5 (Biochemical Methods)

Section cross-reference(s): 2, 13, 14

ST urine diiodothyronine detn excretion; radioimmunoassay diiodothyronine;

thyronine urine excretion; sulfate thyronine excretion; glucuronide thyronine excretion; hyperthyroidism iodothyronine excretion; *hypothyroidism* iodothyronine excretion; thyroid function iodothyronine excretion

IT **Thyroid gland**

(function of, free and conjugated iodothyronines excretion in relation to)

IT **Hyperthyroidism**

Hypothyroidism

(iodothyronine excretion in, free and conjugated)

IT 21462-56-6 29919-72-0 30329-13-6 31135-55-4 64192-57-0
76166-54-6 77074-49-8 79349-15-8 79349-16-9 79349-17-0

RL: ANST (Analytical study)

(excretion of, thyroid function in relation to)

L88 ANSWER 42 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1981:58673 HCAPLUS Full-text

DOCUMENT NUMBER: 94:58673

TITLE: Activation and inactivation of thyroxine by cultured rat hepatoma cells

AUTHOR(S): Sorimachi, Kenji; Niwa, Akira; Yasumura, Yoshihiro

CORPORATE SOURCE: Sch. Med., Dokkyo Univ., Tochigi, 321-02, Japan

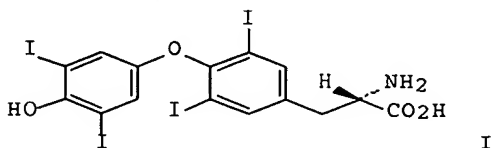
SOURCE: Biochimica et Biophysica Acta, General Subjects (1980), 633(1), 134-43

CODEN: BBGSB3; ISSN: 0304-4165

DOCUMENT TYPE: Journal

LANGUAGE: English

GI



AB The metabolism of thyroxine (I) [51-48-9], 3,3',5-triiodothyronine (II) [6893-02-3], and 3,3',5'-triiodothyronine (III) [5817-39-0] was investigated in rat hepatoma cell cultures. When I was incubated with the cells at 37°, I glucuronide [21462-56-6] was the major product and a little increase in 125I- was detected. Although II was not observed in the incubation medium, II was clearly identified in the EtOH extract obtained from the cell homogenates after 24 h incubation. This cell line also metabolized labeled II added to culture medium. After 24 h incubation, II glucuronide [29919-72-0] was the major metabolite and iodothyronine sulfates were also formed. The sulfates contained II sulfate [31135-55-4] and 3,3'-diiodothyronine sulfate [64192-57-0] and an unknown component. In the metabolism of III, the cells were very active in carrying out glucuronidation and phenolic ring deiodination, and this metabolism yielded III glucuronide [30329-13-6] and 3,3'-diiodothyronine glucuronide [76166-54-6]. The iodide fraction contained a small amount of 3,3'-diiodothyronine sulfate. Thus, these rat hepatoma cells metabolize the thyroid hormones and their analogs by phenolic and nonphenolic ring deiodinations, by glucuronidation, and by sulfation.

CC 2-2 (Hormone **Pharmacology**)

Section cross-reference(s): 14

IT 29919-72-0 31135-55-4 64192-57-0

RL: FORM (Formation, nonpreparative)
(formation of, from T3 by hepatoma culture)

L88 ANSWER 43 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1965:500599 HCAPLUS Full-text

DOCUMENT NUMBER: 63:100599

ORIGINAL REFERENCE NO.: 63:18577f

TITLE: Biosynthesis of *triiodothyronine sulfate* by beef thyroid in vitro

AUTHOR(S): Cohn, George L.

CORPORATE SOURCE: Yale Univ. School of Med.

SOURCE: Nature (London, United Kingdom) (1965), 208(5005), 80

CODEN: NATUAS; ISSN: 0028-0836

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Triiodothyronine sulfokinase (I) activity was investigated in fractionated beef thyroid tissue. I activity is highest in the thyroid microsomal-free supernatant. I activity remains constant for at least 3 months despite frequent freezing and thawing. I activity is also highest in the hepatic and adrenal microsome-free fraction. Beef thyroid microsome-free supernatant synthesizes thyroxine sulfate in the same order of magnitude as *triiodothyronine sulfate*.

CC 58 (Hormones)

IT Thyroid gland

(*triiodothyronine sulfate* formation by, enzyme in)

L88 ANSWER 44 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1964:10995 HCAPLUS Full-text

DOCUMENT NUMBER: 60:10995

ORIGINAL REFERENCE NO.: 60:2006f-h

TITLE: 3,3',5'-Triiodothyronine and 3,3'-diiodothyronine; partially deiodinated intermediates in the metabolism of the thyroid hormones

AUTHOR(S): Flock, Eunice V.; David, Claude; Stobie, George H. C.; Owen, Charles A., Jr.

CORPORATE SOURCE: Mayo Clin., Rochester, MN

SOURCE: Endocrinology (1963), 73(4), 442-55

CODEN: ENDOAO; ISSN: 0013-7227

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB 131-Labeled 3,3',5'-triiodothyronine (I) and 3,3'-diiodothyronine (II) were given to normal and hepatectomized dogs and dogs and rats with biliary fistulas. Radioactivity was determined in bile, urine, and plasma collected from 6 to 24 hrs. following treatment. The fluids were extracted, chromatographed, radioactive spots eluted, hydrolyzed, and chromatographed. Autoradiographs of chromatograms were used to determine the identity and quantity of the metabolic products formed from I and II. In the dog, stepwise deiodination led to the formation of II and 3'-monoiodothyronine (III) from I, and of III from II. These products were found mostly in conjugated form in bile and after hepatectomy in urine. Deiodination from the β -ring occurred more slowly from I and II than from thyroxine or 3,5,3'-triiodothyronine, and was greatly diminished from all these substances after hepatectomy. In the rats a species difference was noted; deiodination from the β -ring of the above compds. was fairly rapid and large amts. of labeled iodide were excreted in the urine. Results indicated that both I and II are important intermediates of a stepwise deamination pathway in the metabolism of thyroxine in the dog, and II is an important intermediate in the metabolism of 3,5,3'-triiodothyronine.

CC 58 (Hormones)

IT Thyroid gland

(hormones of, conjugated metabolites of)

IT 3130-96-9, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-
10056-03-8, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-
diiodophenyl]-, hydrogen sulfate (ester) 21462-56-6, Glucosiduronic
acid, 4-[4-(2-amino-2-carboxyethyl)-2,6-diiodophenoxy]-2,6-diiodophenyl
29919-72-0, Glucosiduronic acid, 4-[4-(2-amino-2-carboxyethyl)-2,6-
diiodophenoxy]-2-iodophenyl 29919-72-0, Alanine, 3-4-4-(glucuronosyloxy)-
3-iodophenoxy]-3,5-diiodophenyl]- 30329-13-6, Glucosiduronic acid,
4-[4-(2-amino-2-carboxyethyl)-2-iodophenoxy]-2,6-diiodophenyl
64192-57-0, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3-iodophenyl]-,
hydrogen sulfate (ester) 64192-58-1, Alanine, 3-[p-(4-hydroxy-3-
iodophenoxy)phenyl]-, hydrogen sulfate (ester) 76166-54-6,
Glucosiduronic acid, 4-[4-(2-amino-2-carboxyethyl)-2-iodophenoxy]-2-
iodophenyl 77074-49-8, Thyroxine, hydrogen sulfate (ester) 77124-61-9,
Glucosiduronic acid, 4-[p-(2-amino-2-carboxyethyl)phenoxy]-2-iodophenyl
77124-61-9, Alanine, 3-[p-[4-(glucuronosyloxy)-3-iodophenoxy]-phenyl]-
79349-15-8, Alanine, 3-[4-(4-hydroxy-3,5-diiodophenoxy)-3-iodophenyl]-,
hydrogen sulfate 100405-48-9, Glucosiduronic acid, 4-[(α -carboxy-2-
iodo-p-tolyl)oxy]-2-iodophenyl 100623-57-2, Glucosiduronic acid,
4-[(α -carboxy-2,6-diiodo-p-tolyl)oxy]-2,6-diiodophenyl
106600-27-5, Glucosiduronic acid, 4-[(α -carboxy-2-iodo-p-tolyl)oxy]-
2,6-diiodophenyl 887229-51-8, Alanine, 3-[4-[4-(glucuronosyloxy)-3-
iodophenoxy]-3-iodophenyl]- 900787-54-4, Acetic acid,
[4-[4-(glucuronosyloxy)-3-iodophenoxy]-3-iodophenyl]- 900787-62-4,
Acetic acid, [4-[4-(glucuronosyloxy)-3,5-diiodophenoxy]-3-iodophenyl]-
900787-66-8, Acetic acid, [4-[4-(glucuronosyloxy)-3,5-diiodophenoxy]-3,5-
diiodophenyl]-

(as thyroid hormone metabolite)

L88 ANSWER 45 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1964:84196 HCAPLUS Full-text

DOCUMENT NUMBER: 60:84196

ORIGINAL REFERENCE NO.: 60:14790g-h

TITLE: Activity of the renal tubule in the excretion of
thyroid hormones

AUTHOR(S): Baschieri, L.; Fabbrini, A.; Mazzuoli, G. F.; Cinotti,
G. A.; Salabe, G. B.; Stirati, G.

CORPORATE SOURCE: Univ. Rome

SOURCE: Minerva Nucleare (1963), 7(10), 389-90

CODEN: MINUA9; ISSN: 0369-0288

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB Using the stop-flow method, dogs were injected with triiodothyronine-131I (I)
or L-thyroxine-131I (II). I did not pass the glomerulus; after its
administration, the urinary chromatograms showed only the inorg. 131I. The
epithelium of the tubule was impermeable to II; the sulfated derivative of II
was absorbed in the distal tubule.

CC 58 (Hormones)

IT *Thyroid gland*

(hormones of, kidney excretion of)

IT 10056-03-8, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-
diiodophenyl]-, hydrogen sulfate (ester)

(resorption of)

L88 ANSWER 46 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1961:88453 HCAPLUS Full-text

DOCUMENT NUMBER: 55:88453

ORIGINAL REFERENCE NO.: 55:16735d-e

TITLE: Nature of the metabolites of thyroid hormones present

in the bile and the plasma of rat, after
administration of radioactive iodides

AUTHOR(S): Gregorio, P. De; Lobo, L. C. G.; Michel, R.; Roche, J.
CORPORATE SOURCE: College of France, Paris
SOURCE: Bulletin de la Societe de Chimie Biologique (1960),
42, 1213-21
CODEN: BSCIA3; ISSN: 0037-9042

DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB cf. CA 52, 20622c. Glucuroconjugates of thyroxine, of 3,3',5-triiodothyronine
and of 3,3',5,5'-tetraiodothyroacetic acid, and sulfoconjugates of the same
compds. and of 3,3',5-triiodothyroacetic acid were detected in the bile of
rats receiving repeated injections of very low and fractionated doses of
NaI131. All these compds. are products of the normal metabolism of thyroid
hormones.

CC 11F (Biological Chemistry: Physiology)

IT *Thyroid gland*
(hormones of, deiodases in metabolism of)

IT *Thyroid gland*
(hormones of, metabolites of, in bile and blood plasma)

IT 10056-03-8, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-
diiodophenyl]-, H sulfate 95786-11-1, Acetic acid, [4-(4-hydroxy-3-
iodophenoxy)-3,5-diiodophenyl]-, H sulfate 108950-89-6, Acetic acid,
[4-(4-hydroxy-3,5-diiodophenoxy)-3,5-diiodophenyl]-, H sulfate
885456-76-8, Acetic acid, [4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-,
glucuronate 887229-11-0, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-
diiodophenyl]-, glucuronate 896440-75-8, Acetic acid,
[4-(4-hydroxy-3,5-diiodophenoxy)-3,5-diiodophenyl]-, glucuronate
(formation in bile and blood plasma, I131 effect on)

L88 ANSWER 47 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1961:44511 HCAPLUS Full-text
DOCUMENT NUMBER: 55:44511
ORIGINAL REFERENCE NO.: 55:8638h-i
TITLE: Further study of the degradation of
3,3',5-triiodo-L-thyronine sulfuric ester by rat feces

AUTHOR(S): Closon, Jacques; Betz-Bareau, M.
CORPORATE SOURCE: Univ. Liege, Belg.
SOURCE: Comptes Rendus des Seances de la Societe de Biologie
et de Ses Filiales (1960), 154, 1109-12
CODEN: CRSBAW; ISSN: 0037-9026

DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB cf. CA 55, 2787g. Hydrolysis of the ester at neutral pH and 37° by a
suspension of rat feces was not due to Escherichia coli, but partly to other
unidentified bacteria and partly to an exosulfatase of uncertain bacterial
origin. The subsequent deamination, after hydrolysis of the ester, was by E.
coli and not by an exoenzyme in the medium.

CC 11H (Biological Chemistry: *Pharmacology*)

IT 31135-55-4, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-
diiodophenyl]-, sulfate (ester) 31135-55-4, Sulfuric acid, ester
with 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]alanine
(hydrolysis by feces)

L88 ANSWER 48 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1960:82202 HCAPLUS Full-text
DOCUMENT NUMBER: 54:82202
ORIGINAL REFERENCE NO.: 54:15705c-e
TITLE: Metabolism in the rat of the sulfuric ester of
3,3',5-triiodothyronine

AUTHOR(S): Roche, Jean; Michel, Raymond; Closon, Jacques; Michel, Odette
CORPORATE SOURCE: College of France, Paris
SOURCE: Biochimica et Biophysica Acta (1960), 38, 325-32
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: French
AB cf. CA 53, 19087h. The metabolism of 3,3',5-triiodothyronine (I) and its sulfuric ester (II) was studied in the thyroidectomized rat. I was degraded much faster than II, which the cells concentrated to a much lesser degree. The characteristics of the metabolism of II, including its formation at the expense of the glucuronoconjugate of I in the liver and the excretion of I after its injection, its presence in plasma, and its absence from the urine, suggested that it could be hydrolyzed in the tissues by an arylsulfatase and its hormonal component deiodinated. It is suggested that the probable role of II is to serve as a reserve of I accessible to the cells after hydrolysis of the ester bond.
CC 11H (Biological Chemistry: *Pharmacology*)
IT 31135-55-4, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-, sulfate (ester) 31135-55-4, Sulfuric acid, ester with 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]alanine (metabolism of)

L88 ANSWER 49 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1959:57904 HCAPLUS Full-text
DOCUMENT NUMBER: 53:57904
ORIGINAL REFERENCE NO.: 53:10517b-c
TITLE: Biliary excretion of the sulfoconjugate of 3,3',5-triiodothyronine after injection of thyroxine
AUTHOR(S): Roche, Jean; Michel, Raymond; Gruson, Marcelle
CORPORATE SOURCE: College of France, Paris
SOURCE: Comptes Rendus des Seances de la Societe de Biologie et de Ses Filiales (1958), 152, 1324-8
CODEN: CRSBAW; ISSN: 0037-9026
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Bile of thyroidectomized rats contained small amts. of sulfoconjugated 3,3',5-triiodo-L-thyronine after i.m. injection of DL-thyroxine.
CC 11H (Biological Chemistry: *Pharmacology*)
IT Bile (3,3',5-triiodothyronine sulfate in)
IT 31135-55-4, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-, sulfate (ester) 31135-55-4, Sulfuric acid, ester with 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl] alanine (in bile after thyroxine administration)

L88 ANSWER 50 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1958:88973 HCAPLUS Full-text
DOCUMENT NUMBER: 52:88973
ORIGINAL REFERENCE NO.: 52:15693h-i
TITLE: A sulfate ester of 3,5,3'-triiodo-L-thyronine in human plasma and bile
AUTHOR(S): Fauvert, R.; Roche, J.; Michel, R.; Thieblemont, P.; Gruson, M.
CORPORATE SOURCE: Coll. France, Paris
SOURCE: Rev. franc. etudes clin. et biol. (1958), 3, 372-4
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB A sulfate ester of 3,5,3'-triiodo-L-thyronine was found in plasma and bile following the injection of labelled 3,5,3'-triiodo-L-thyronine in *hypothyroid* patients.
CC 11F (Biological Chemistry: Physiology)
IT 31135-55-4, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-, sulfate (ester)
(in bile and blood plasma)

L88 ANSWER 51 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1958:116115 HCAPLUS Full-text
DOCUMENT NUMBER: 52:116115
ORIGINAL REFERENCE NO.: 52:20622d-e
TITLE: Biliary, urinary, and fecal elimination and tissue distribution of 3,3',5-triiodothyronine and its sulfuric ester in the rat
AUTHOR(S): Roche, Jean; Michel, Raymond; Closon, Jacques; Michel, Odette
CORPORATE SOURCE: Coll. France, Paris
SOURCE: Comptes Rendus des Seances de la Societe de Biologie et de Ses Filiales (1958), 152, 33-8
CODEN: CRSBAW; ISSN: 0037-9026
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB I131-labeled I and II were injected intravenously into thyroidectomized rats. Diffusion into the tissues was slower for II than for I. Both were partly dehalogenated in the body. Over half of the radioactivity was excreted in 4 hrs. in the urine (.apprx.41%) and feces (.apprx.13%). That in the urine was mostly inorg. iodide. No II was excreted unchanged in the urine. Some I and II passed into the bile.

CC 11H (Biological Chemistry: *Pharmacology*)

IT *Thyroidectomized state*

Thyroidectomized state

(3,3',5-triiodothyronine and its sulfuric esters in bile, feces, tissue and urine in)

IT 3130-96-9, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-
31135-55-4, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-, sulfate (ester)
(in bile, feces, tissue and urine after thyroidectomy)

L88 ANSWER 52 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1958:116114 HCAPLUS Full-text
DOCUMENT NUMBER: 52:116114
ORIGINAL REFERENCE NO.: 52:20622c-d
TITLE: Presence of the sulfuric ester of 3,3',5-triiodo-L-thyronine in the plasma of the rat after administration of the hormone
AUTHOR(S): Roche, Jean; Michel, Raymond; Closon, Jacques; Michel, Odette
CORPORATE SOURCE: Coll. France, Paris
SOURCE: Comptes Rendus des Seances de la Societe de Biologie et de Ses Filiales (1958), 152, 6-10
CODEN: CRSBAW; ISSN: 0037-9026
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Within a few hrs. after intramuscular injection of 3,3',5-triiodothyronine (I) into thyroidectomized rats its sulfoconjugate (II) was found present in small amts. in the circulating blood. II should be regarded as a normal plasma constituent. The isolation and identification of II are described.

CC 11H (Biological Chemistry: *Pharmacology*)

IT 31135-55-4, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-

diiodophenyl]-, sulfate (ester)
(in blood plasma after administration)

L88 ANSWER 53 OF 63 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1958:26607 HCAPLUS Full-text

DOCUMENT NUMBER: 52:26607

ORIGINAL REFERENCE NO.: 52:4833e-f

TITLE: Biliary excretion of a sulfoconjugate of
3,5,3'-triiodo-L-thyronine after administration of
that hormone to a rat

AUTHOR(S): Roche, Jean; Michel, Raymond; Michel, Odette; Etling,
Nicole

SOURCE: Compt. rend. (1957), 245, 1089-91

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB Male thyroidectomized rats were given s.c. 3,5,3'-triiodo-L-thyronine (I)
labeled with I131 in the 3' position and with or without S35-labeled Na2SO4.
The bile collected after injection was fractionated by means of chromatog.
The two principal marked compds. determined after I administration were I and
the sulfuric ester of I. This compound (II) was also identified in the blood.
Chromatog. studies also revealed the radioactive constituents: SO4--;
glycuroconjugate of I; triiodothyropyruvic acid; II; and an unknown
metabolite.

CC 11H (Biological Chemistry: *Pharmacology*)

IT 6893-02-3, Alanine, 3-[4-(4-hydroxy-3-iodophenoxy)-3,5-diiodophenyl]-,
glucuronoside 31135-55-4, Alanine, 3-[4-(4-hydroxy-3-
iodophenoxy)-3,5-diiodophenyl]-, sulfate (ester)
(biliary excretion after administration)

L88 ANSWER 54 OF 63 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 96434364 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 8837331

TITLE: Demonstration of thyromimetic effects of 3,5,3'-
triiodothyronine sulfate (T3S) in
euthyroid rats.

AUTHOR: Chopra I J; Nguyen D

CORPORATE SOURCE: Department of Medicine, UCLA School of Medicine 90024, USA.

SOURCE: Thyroid : official journal of the American Thyroid
Association, (1996 Jun) Vol. 6, No. 3, pp. 229-32.
Journal code: 9104317. ISSN: 1050-7256.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199612

ENTRY DATE: Entered STN: 28 Jan 1997

Last Updated on STN: 28 Jan 1997

Entered Medline: 12 Dec 1996

ABSTRACT:

We have previously demonstrated that T3 sulfate (T3S) exhibits thyromimetic
effects in *hypothyroid* rats and that, on a molar basis, its activity
approximates 20% that of T3. Since T3S is avidly deiodinated by 5'-deiodinase,
type I (5'-DI) and 5'-DI activity is markedly reduced in *hypothyroidism*
, it seemed possible that T3S is active only in *hypothyroidism* and
not in the euthyroid state wherein normal tissue 5'-DI activity will rapidly
degrade T3S and little T3S will be left for metabolism (desulfation) to
biologically active T3. This study was undertaken to test this possibility.

We studied the effect of T3S (4.6, 14, or 42 nmol/day for 7 days, ip) and T3 (1.0, 3.0 or 9.0 nmol/day for 7 days, ip) in groups of male Sprague-Dawley rats (5-6/group); the control group was treated with saline ip. Treatment with both T3 and T3S caused a significant ($p < 0.05$) increase in hepatic and renal 5'-DI. Similarly, both treatments caused a significant reduction in serum total T4 and TSH levels. In these effects, T3S was approximately one-fifth as potent as T3 on a molar basis. Interestingly, changes in body weight during treatment with T3 and T3S suggested that at doses that caused a comparable tissue or pituitary effect, T3S treatment permitted a significantly greater weight gain than treatment with T3. We conclude that T3S exhibits thyromimetic effects in euthyroid rats in a manner comparable to that in *hypothyroid* rats. The biological effects of T3S may be due to T3 generated in tissues by desulfation of T3S.

CONTROLLED TERM: Check Tags: Male

Animals

*Iodide Peroxidase: ME, metabolism

Kidney: DE, drug effects

Kidney: EN, enzymology

Liver: DE, drug effects

Liver: EN, enzymology

Rats

Rats, Sprague-Dawley

Research Support, Non-U.S. Gov't

Thyrotropin: BL, blood

Thyroxine: BL, blood

*Triiodothyronine: AA, analogs & derivatives

Triiodothyronine: PD, pharmacology

CAS REGISTRY NO.: 31135-55-4 (triiodothyronine sulfate); 6893-02-3 (Triiodothyronine); 7488-70-2 (Thyroxine); 9002-71-5 (Thyrotropin)

CHEMICAL NAME: EC 1.11.1.8 (Iodide Peroxidase)

L88 ANSWER 55 OF 63

MEDLINE on STN

DUPLICATE 8

ACCESSION NUMBER: 93273855 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 8501166

TITLE: A study of the serum 3,5,3'-triiodothyronine sulfate concentration in normal and *hypothyroid* fetuses at various gestational stages.

AUTHOR: Santini F; Cortelazzi D; Baggiani A M; Marconi A M; Beck-Peccoz P; Chopra I J

CORPORATE SOURCE: Department of Medicine, University of California, Los Angeles 90024.

CONTRACT NUMBER: DK-16155 (NIDDK)

NSS2-S07-RR-05354 (NCRR)

SOURCE: The Journal of clinical endocrinology and metabolism, (1993 Jun) Vol. 76, No. 6, pp. 1583-7.

Journal code: 0375362. ISSN: 0021-972X.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199307

ENTRY DATE: Entered STN: 16 Jul 1993

Last Updated on STN: 29 Jan 1996

Entered Medline: 1 Jul 1993

ABSTRACT:

We have studied T3 sulfate (T3S) levels, blindly, in coded plasma samples from 21 normal and 3 *hypothyroid* fetuses at different stages of gestation (19-42 weeks). Fetal plasma samples were obtained by cordocentesis. T3S was detectable in all samples studied, with values ranging from 50-294 (mean +/-

SD, 130 +/- 62 pmol/L). Plasma T3S was low (< 45 pmol/L) in all 4 normal adult control subjects studied simultaneously; serum T3S ranged from less than 20 to 130 in another set of 18 control subjects (mean +/- SD, 63 +/- 32 pmol/L). Fetal T3S values were positively correlated with gestational age ($r = 0.43$; $P < 0.05$), but not with free T4 (FT4), FT3, or TSH values. In the 3 ***hypothyroid*** fetuses at 31, 38, and 40 weeks gestation, respectively, plasma TSH was elevated (26, 98, and 24 mU/L, respectively), FT4 was low (10, 6.7, and 7.5 pmol/L, respectively), and FT3 was normal or high (3.2, 8.2, and 2.2 pmol/L, respectively). However, T3S values in *hypothyroid* fetuses (88, 133, and 252 pmol/L, respectively) were similar to those in normal fetuses at corresponding gestational ages. We conclude that 1) T3S is detectable in fetal circulation from at least 19 weeks' gestation, and its concentration increases with fetal-age; 2) plasma T3S concentrations in the fetus at 19-40 weeks gestation are at least comparable to but generally higher than those in the adult; and 3) plasma T3S levels in *hypothyroid* fetuses are similar to those in normal fetuses. Recent studies demonstrating the ability of some fetal rat tissues (e.g. cerebral cortex) to desulfate T3S to T3 have suggested a possible role of T3S as a source of T3. Normal T3S in fetal *hypothyroidism* suggests that T3S may contribute to attenuation of the effects of *hypothyroidism* during intrauterine life.

CONTROLLED TERM: Check Tags: Female; Male
 Embryonic and Fetal Development
 *Fetal Diseases: BL, blood
 Gestational Age
 Humans
 *Hypothyroidism: BL, blood
 Osmolar Concentration
 Reference Values
 Research Support, Non-U.S. Gov't
 Research Support, U.S. Gov't, P.H.S.
 Thyrotropin: BL, blood
 Thyroxine: BL, blood
 *Triiodothyronine: AA, analogs & derivatives
 Triiodothyronine: BL, blood

CAS REGISTRY NO.: 31135-55-4 (*triiodothyronine sulfate*); 6893-02-3
 (Triiodothyronine); 7488-70-2 (Thyroxine); 9002-71-5
 (Thyrotropin)

L88 ANSWER 56 OF 63 MEDLINE on STN DUPLICATE 11
 ACCESSION NUMBER: 92317293 MEDLINE Full-text
 DOCUMENT NUMBER: PubMed ID: 1619009
 TITLE: A radioimmunoassay for measurement of 3,5,3'-
triiodothyronine sulfate: studies in
thyroidal and nonthyroidal *diseases*,
 pregnancy, and neonatal life.
 AUTHOR: Chopra I J; Wu S Y; Tecu G N; Santini F
 CORPORATE SOURCE: Department of Medicine, University of California-Los
 Angeles Center for Health Sciences 90024-1682.
 CONTRACT NUMBER: DK-16155 (NIDDK)
 SOURCE: The Journal of clinical endocrinology and metabolism, (1992
 Jul) Vol. 75, No. 1, pp. 189-94.
 Journal code: 0375362. ISSN: 0021-972X.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 199207
 ENTRY DATE: Entered STN: 15 Aug 1992
 Last Updated on STN: 15 Aug 1992
 Entered Medline: 31 Jul 1992

ABSTRACT:

A highly sensitive, specific, and reproducible RIA has been developed to measure T3 sulfate (T3S). Only T4 sulfate cross-reacted significantly (approximately 3%) in the RIA; rT3 sulfate, T4, T3, rT3, and diiodothyronine cross-reacted less than 0.01%. T3S was bound by thyronine-binding globulin and albumin in serum. The free fraction of T3S in four normal sera averaged 0.25% compared to a value of 0.35% for T3. Therefore, T3S was measured in ethanol extracts of serum. Recovery of the nonradioactive T3S added to serum averaged 92%. The dose-response curves of inhibition of binding of [125I]T3S to anti-T3S antibody by serial dilutions of serum extracts were essentially parallel to the standard curve. The detection threshold of the RIA was 20 pmol/L (1.5 ng/dL). The coefficient of variation averaged 7.8% within an assay and 11% between assays. The serum concentration of T3S was (mean \pm SE) 76 \pm 7.2 pmol/L in normal subjects, 268 \pm 29 in hyperthyroid patients with Graves' disease, 92 \pm 28 in *hypothyroid* patients, 201 \pm 32 in patients with systemic nonthyroidal illnesses, 40 \pm 6.2 in pregnant women (15-31 weeks gestation), and 429 \pm 39 in cord sera of newborns; the values in hyperthyroidism, nonthyroidal illnesses, and newborns were significantly different from normal (P less than 0.01). The mean concentration of T3S in amniotic fluid samples at 15-31 weeks gestation (90 \pm 1.3 pmol/L) was significantly higher than the corresponding value in maternal serum (P less than 0.05) and significantly lower than the corresponding value in newborn cord blood serum (P less than 0.001). Oral administration of sodium ipodate (Oragrafin; 3 g) to two hyperthyroid patients was associated with a 76-190% increase in serum T3S at 8 h, followed by a gradual decrease to a nadir that was 25-60% of the baseline value 2-3 days after ipodate ingestion. We conclude that 1) T3S is a normal component of human serum, and its levels change substantially in several physiological and pathological conditions; 2) sulfation pathway plays an important role in the metabolism of iodothyronines in man; and 3) high serum T3S levels in newborns and low normal levels in pregnancy despite elevated thyronine-binding globulin levels may signify markedly different metabolism of T3S in the mother and fetus.

CONTROLLED TERM:

Check Tags: Female
Adolescent
Adult
Aged
Amniotic Fluid: CH, chemistry
Carrier Proteins: BL, blood
Dose-Response Relationship, Drug
Fetal Blood: CH, chemistry
Graves Disease: BL, blood
Humans
Infant, Newborn
Membrane Proteins: BL, blood
Middle Aged
Pregnancy: BL, blood
*Radioimmunoassay: MT, methods
Reference Standards
Reproducibility of Results
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
*Thyroid Hormones
*Triiodothyronine: AA, analogs & derivatives
Triiodothyronine: AN, analysis
Triiodothyronine: BL, blood

CAS REGISTRY NO.: 31135-55-4 (triiodothyronine sulfate); 6893-02-3
(Triiodothyronine)

CHEMICAL NAME: 0 (Carrier Proteins); 0 (Membrane Proteins); 0 (Thyroid Hormones); 0 (thyroid hormone-binding proteins)

L88 ANSWER 57 OF 63 MEDLINE on STN

ACCESSION NUMBER: 2000035056 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 10567020

TITLE: Fetal-to-maternal transfer of 3,3',5-triiodothyronine sulfate and its metabolite in sheep.

AUTHOR: Wu S Y; Polk D H; Huang W S; Fisher D A

CORPORATE SOURCE: Nuclear Medicine Services, Department of Veterans Affairs Medical Center, Long Beach, California 90822, USA..
sywu@pop.long-beach.va.gov

CONTRACT NUMBER: HD-04270 (NICHD)
R15-GM-41949 (NIGMS)

SOURCE: The American journal of physiology, (1999 Nov) Vol. 277, No. 5 Pt 1, pp. E915-9.
Journal code: 0370511. ISSN: 0002-9513.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199912

ENTRY DATE: Entered STN: 13 Jan 2000

Last Updated on STN: 13 Jan 2000

Entered Medline: 14 Dec 1999

ABSTRACT:

Earlier studies have shown that sulfoconjugation is a major pathway of thyroid hormone metabolism in fetal mammals. To assess the placental transfer of sulfoconjugates in the pregnant sheep model, we measured 3,3',5-triiodothyronine (T(3)) sulfate (T(3)S), 3,3'-diiodothyronine sulfate (T(2)S), and T(3) concentrations in fetal serum and in maternal serum and urine after T(3)S infusion to the fetus (n = 5) or the ewe (n = 6). Maternal infusion of T(3)S did not increase fetal serum T(2)S, T(3)S, or T(3) concentrations. In contrast, fetal infusion of T(3)S produced significant increases in maternal serum T(2)S and T(3)S but not T(3) concentrations. Fetal T(3)S infusion also increased maternal urine excretion of T(3)S. However, the 4-h cumulative maternal urinary excretion of T(2)S and T(3)S after fetal T(3)S infusion was less than the excretion observed after fetal infusion of equimolar amounts of T(3) in our previous study. It is concluded that fetal serum T(2)S and T(3)S can be transferred to maternal compartments. However, compared with T(3), these sulfoconjugates may be less readily transferred.

CONTROLLED TERM: Check Tags: Female

Animals

Diiodothyronines: BL, blood

**Diiodothyronines: PK, pharmacokinetics*

Diiodothyronines: UR, urine

Fetus: ME, metabolism

**Maternal-Fetal Exchange: PH, physiology*

Pregnancy

Research Support, Non-U.S. Gov't

Research Support, U.S. Gov't, Non-P.H.S.

Research Support, U.S. Gov't, P.H.S.

Sheep

Thyroid Gland: PH, physiology

**Triiodothyronine: AA, analogs & derivatives*

Triiodothyronine: BL, blood

Triiodothyronine: PK, pharmacokinetics

Triiodothyronine: UR, urine

CAS REGISTRY NO.: 31135-55-4 (triiodothyronine sulfate); 64192-57-0
(3,3'-diiodothyronine-4-sulfate); 6893-02-3
(Triiodothyronine)

CHEMICAL NAME: 0 (Diiodothyronines)

ACCESSION NUMBER: 97374603 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 9231052
TITLE: Triiodothyronine (T3) reflects renal graft function after renal transplantation.
AUTHOR: Reinhardt W; Misch C; Jockenhovel F; Wu S Y; Chopra I; Philipp T; Reinwein D; Eigler F W; Mann K
CORPORATE SOURCE: Division of Endocrinology, Medical Clinic, Essen, Germany.
SOURCE: Clinical endocrinology, (1997 May) Vol. 46, No. 5, pp. 563-9.
Journal code: 0346653. ISSN: 0300-0664.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 25 Aug 1997
Last Updated on STN: 25 Aug 1997
Entered Medline: 11 Aug 1997

ABSTRACT:

OBJECTIVE: Abnormalities in *thyroid* function are observed in patients with end stage renal *disease*. However, there are no data available evaluating sequential changes of *thyroid* function after renal transplantation. Therefore, we have studied *thyroid* hormone function in the immediate post-operative period after renal transplantation in order to determine the relationship between improving renal function and changes in ****thyroid**** hormone economy. DESIGN AND PATIENTS: *Thyroid* function was evaluated in 22 patients before and on days 1, 3, 7 and 15 after renal transplantation. All patients received prednisone and cyclosporin as immunosuppressive therapy. Twelve patients with normal renal function undergoing comparable surgical procedures served as a control group. MEASUREMENTS: Serum creatinine and *thyroid* hormone parameters (total T4, total T3, free T4, free T3, thyroxin binding globulin (TBG), reverse T3, T3 sulphate and TSH) were measured. RESULTS: According to post-operative kidney function after renal transplantation, patients could be subdivided into three groups: five patients had primary graft function (group I); seven patients had delayed graft function because of acute renal failure (group II); 10 patients had delayed graft function requiring high doses of prednisone and some also of OKT3 because of acute rejection (group III). There was a significant fall in T3 and T4 concentrations with a concomitant rise in reverse T3 in all patients up to 3 days after renal transplantation. However, only patients in group I reached pre-operative values on day 15 after renal transplantation (serum creatinine 167 +/- 52 micromM), whereas patients in group II (creatinine 609 +/- 118 micromM) and group III (creatinine 839 +/- 71 micromM) continued to have T3 concentrations well in the *hypothyroid* range (group I, 1.68 +/- 0.28 nM) vs 0.87 +/- 0.09 nM in group II and 0.76 +/- 0.10 nM in group III; P < 0.01). Serum T4 concentrations were also low in group III (47.7 nM vs 100.2 nM in group I; P < 0.05) 15 days after renal transplantation. These changes were accompanied by a concomitant fall in T3/TBG ratio and in free T3. Elevated reverse T3 returned to normal values in all groups on the 15th day after renal transplantation. TSH fell significantly on the first post-operative day, but did not return to pre-operative values in renal transplantation patients. In the control group, TSH did not change during the study period. T3 sulphate, known to be elevated in chronic renal failure, remained above normal in all patients irrespective of graft function during this study period. CONCLUSIONS: T3 concentrations reflect renal graft function after renal transplantation. T3 is below normal in patients with delayed graft function (acute renal failure or acute rejection). The post-operative period (up to 3 days after renal transplantation) is associated with a low T3 syndrome. TSH does not return to

pre-operative values even in patients with primary graft function. This might be due to the administration of prednisone. T3-sulphate is elevated before and after renal transplantation irrespective of graft function.

CONTROLLED TERM: Check Tags: Female; Male
Adult
Biological Markers: BL, blood
Cyclosporine: TU, therapeutic use
Graft Rejection: BL, blood
*Graft Rejection: DI, diagnosis
Graft Rejection: PP, physiopathology
Humans
Immunosuppressive Agents
Kidney: PP, physiopathology
Kidney Failure, Acute: BL, blood
*Kidney Failure, Acute: DI, diagnosis
Kidney Failure, Acute: PP, physiopathology
*Kidney Transplantation
Middle Aged
Postoperative Period
Prednisolone: TU, therapeutic use
Thyroid Gland: PP, physiopathology
Thyrotropin: BL, blood
Thyroxine: BL, blood
Thyroxine-Binding Proteins: AN, analysis
Triiodothyronine: AA, analogs & derivatives
*Triiodothyronine: BL, blood
Triiodothyronine, Reverse: BL, blood

CAS REGISTRY NO.: 31135-55-4 (*triiodothyronine sulfate*); 50-24-8
(Prednisolone); 5817-39-0 (Triiodothyronine, Reverse);
59865-13-3 (Cyclosporine); 6893-02-3 (Triiodothyronine);
7488-70-2 (Thyroxine); 9002-71-5 (Thyrotropin)
CHEMICAL NAME: 0 (Biological Markers); 0 (Immunosuppressive Agents); 0
(Thyroxine-Binding Proteins)

L88 ANSWER 59 OF 63 MEDLINE on STN
ACCESSION NUMBER: 96314737 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 8733878
TITLE: Increased urinary excretion of sulfated
3,3',5-triiodothyronine in patients with nodular goiters
receiving suppressive thyroxine therapy.
AUTHOR: Huang W S; Kuo S W; Chen W L; Fuh M M; Wu S Y
CORPORATE SOURCE: Department of Nuclear Medicine, Tri-Service General
Hospital, Taipei, Taiwan, R.O.C.
CONTRACT NUMBER: HD 04270 (NICHD)
R15-GM 41949 (NIGMS)
SOURCE: Thyroid : official journal of the American Thyroid
Association, (1996 Apr) Vol. 6, No. 2, pp. 91-6.
Journal code: 9104317. ISSN: 1050-7256.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 6 Nov 1996
Last Updated on STN: 6 Nov 1996
Entered Medline: 24 Oct 1996

ABSTRACT:
Increased serum 3,3',5-triiodothyronine sulfate (T3S)
levels have been detected in various pathophysiologic states. However, little

is known about T3S concentrations in other biological fluids. By employing a highly sensitive, specific, and reproducible radioimmunoassay (RIA), we measured T3S in the serum and urine of 20 premenopausal women with benign nodular goiters before and after administration of thyroxine for 6 months (T4; 3.2 micrograms/kg/day). Serum T3 concentrations did not change significantly after treatment (2.0 vs. 1.7 nmol/L; $p > 0.05$). However, the mean serum T4 and free T4 concentrations were significantly higher after treatment (138 vs. 88 nmol/L and 28 vs. 17 pmol/L; $p < 0.01$, respectively). Serum thyroid stimulating hormone (TSH) levels were significantly reduced after T4 treatment (0.13 vs. 0.66 mU/L, $p < 0.01$) and the serum levels of T3S were significantly increased after treatment (82 vs. 45 pmol/L; $p < 0.01$). A good correlation was observed between increased serum T3S and T4 concentrations ($r = 0.66$; $p < 0.001$). The sulfoconjugate of T3 was significantly increased in creatinine-corrected urine after treatment (606 vs. 253 pmol/umol Cr.; $p < 0.01$). There was a significant correlation between increased creatinine-corrected urine T3S and increased serum free T4 ($r = 0.65$; $p < 0.001$). In summary, significant increases in serum and urine T3S levels were noted in T4-treated patients with subnormal serum TSH and borderline elevated T4. We thus conclude that the sulfation pathway may play a role in the homeostasis of thyroid hormone metabolism in T4-treated subjects with relative hyperthyroxinemia. In addition, the creatinine-corrected urine concentrations of T3S may serve as an index for the evaluation of T4-treated patients with elevated levels of T4.

CONTROLLED TERM: Check Tags: Female
Adult
Creatinine: UR, urine
Goiter, Nodular: DT, drug therapy
*Goiter, Nodular: UR, urine
Humans
Iodine Radioisotopes: DU, diagnostic use
Middle Aged
Radioimmunoassay
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Thyrotropin: BL, blood
*Thyroxine: AE, adverse effects
Thyroxine: BL, blood
Thyroxine: TU, therapeutic use
*Triiodothyronine, Reverse: UR, urine
CAS REGISTRY NO.: 5817-39-0 (Triiodothyronine, Reverse); 60-27-5
(Creatinine); 7488-70-2 (Thyroxine); 9002-71-5
(Thyrotropin)
CHEMICAL NAME: 0 (Iodine Radioisotopes)

L88 ANSWER 60 OF 63 MEDLINE on STN
ACCESSION NUMBER: 91365818 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 1890147
TITLE: Characteristics of 3,5,3'-*triiodothyronine*
sulfate metabolism in euthyroid man.
AUTHOR: LoPresti J S; Mizuno L; Nimalysuria A; Anderson K P;
Spencer C A; Nicoloff J T
CORPORATE SOURCE: University of Southern California, School of Medicine,
Department of Medicine, Los Angeles 90033.
CONTRACT NUMBER: DK-11727 (NIDDK)
M01-RR-43 (NCRR)
SOURCE: The Journal of clinical endocrinology and metabolism, (1991
Oct) Vol. 73, No. 4, pp. 703-9.
Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 3 Nov 1991
Last Updated on STN: 3 Nov 1991
Entered Medline: 17 Oct 1991

ABSTRACT:

The sulfated conjugate of T3 (T3S) has long been recognized as a normal product of peripheral thyroid hormone metabolism. In order to better understand the role that T3S may play in this process, the metabolic handling of T3S was studied in euthyroid man. After the iv administration of [¹²⁵I]T3S in man, T3S was found to be rapidly metabolized with estimated mean MCR of 135 +/- 15 liters/day (L/D) after a bolus injection and 127 +/- 8 L/D employing a constant infusion. The primary route of T3S disposal was by deiodination with an efficiency of 92%. The administration of propylthiouracil (PTU, 300 mg every 6 h x 5 days) and iopanoic acid (IA, 500 mg every day x 5 days), both inhibitors of deiodination, decreased clearance compared to control (87 +/- 9 L/D, P less than 0.01 and 46 +/- 10 L/D, P less than 0.002, respectively). A 3-day fast also reduced the clearance of T3S (56 +/- 10 L/D, P less than 0.002). All three maneuvers decreased the total urinary deiodination fraction of tracer T3S (control 91 +/- 2%, PTU 70 +/- 9%, P less than 0.04, IA 26 +/- 3%, P less than 0.0001, and fasting 58 +/- 6%, P less than 0.01). A strong correlation between T3S clearance and deiodination was noted for fasting and IA only (r = 0.78, P less than 0.003). However, no relationship between clearance and deiodination was noted with PTU administration presumably as a result of a compensatory increase in biliary losses of T3S. The urinary thyronine excretion pattern demonstrated the presence of small amounts of labeled T3,3,3'-T2, and 3,3'-T2S with the major metabolite being T3S itself. TSH levels were not influenced by the infusion of stable T3S designed to achieve a serum value greater than 50 ng/dL. No absorption of intact T3S was detected after its oral ingestion. In conclusion, T3S is rapidly cleared from the serum, primarily by deiodination, may undergo nondeiodinative disposal when hepatic deiodination is inhibited by PTU but not with IA or fasting, and has no intrinsic biological activity. Thus, T3S may serve as a metabolite of T3 for its rapid deiodinative disposal. Although the precise role T3S plays in human thyroid hormone metabolism has not been defined, the metabolic characteristics of T3S appear similar to that of an unidentified alternate T4 metabolite formed in low T3 states of fasting and nonthyroidal illness.

CONTROLLED TERM: Check Tags: Male
Administration, Oral
Adult
Fasting: ME, metabolism
Humans
Injections, Intravenous
Iodine Radioisotopes: DU, diagnostic use
Iodine Radioisotopes: ME, metabolism
Iodine Radioisotopes: PK, pharmacokinetics
Middle Aged
Research Support, U.S. Gov't, P.H.S.
Thyroid Gland: ME, metabolism
Triiodothyronine: AD, administration & dosage
*Triiodothyronine: AA, analogs & derivatives
Triiodothyronine: ME, metabolism
Triiodothyronine: PK, pharmacokinetics
CAS REGISTRY NO.: 31135-55-4 (triiodothyronine sulfate); 6893-02-3
(Triiodothyronine)
CHEMICAL NAME: 0 (Iodine Radioisotopes)

reserved on STN

ACCESSION NUMBER: 93226368 EMBASE Full-text
DOCUMENT NUMBER: 1993226368
TITLE: Sulfate conjugates of iodothyronines in developing sheep:
Effect of fetal *hypothyroidism*.
AUTHOR: Wu S.-Y.; Polk D.H.; Huang W.-S.; Reviczky A.; Wang K.;
Fisher D.A.
CORPORATE SOURCE: Nuclear Medicine/Medical Services, VA Medical Center, Long
Beach, CA 90822, United States
SOURCE: American Journal of Physiology - Endocrinology and
Metabolism, (1993) Vol. 265, No. 1 28-1, pp. E115-E120. .
ISSN: 0002-9513 CODEN: AJPM
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
003 Endocrinology
021 Developmental Biology and Teratology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Sep 1993
Last Updated on STN: 12 Sep 1993
ABSTRACT: We recently showed that thyroxine sulfate (T4S) and 3,3',5-
triiodothyronine sulfate (T3S) were major thyroid hormone
metabolites in ovine fetuses and neonates. To further characterize the
sulfation pathway in ovine fetuses, we measured 3,3',5'-triiodothyronine (rT3S)
in serum and other body fluids in samples obtained from fetal (n = 23, 94-145
days of gestational age, term = 150 days), newborn (n = 6), and adult (n = 6)
sheep. In addition, T3S, T4S, and rT3S levels were measured in tissue fluids
and serum samples obtained from ovine fetuses 13 days after total
thyroidectomy (Tx) conducted at gestational age of 110-113 days (n =
5). Sham-operated twin fetuses served as controls (n = 5). The relative order
of mean rT3S concentration for various tissue fluids in fetuses were meconium >
bile > serum > allantoic fluid > urine or amniotic fluid. Peak mean tissue
fluid levels generally occurred at 110-130 days gestation. In
hypothyroid fetuses, significant decreases in the mean serum
concentrations of T4S and rT3S, but not T3S, were noted. The mean rT3S level
also was decreased significantly in allantoic fluid, bile, and meconium,
whereas T4S and T3S levels were reduced only in bile of the Tx fetuses. These
data demonstrate that sulfation is a major pathway in thyroid hormone
metabolism in both euthyroid and *hypothyroid* ovine fetuses.
CONTROLLED TERM: Medical Descriptors:
**hypothyroidism*
*sulfation
*thyroid hormone metabolism
adolescent
age
animal experiment
animal model
animal tissue
article
body fluid
conjugate
controlled study
fetus
newborn
nonhuman
priority journal
serum
sheep

thyroidectomy

tissue distribution

Drug Descriptors:

*iodothyronine: EC, endogenous compound

*liothyronine: EC, endogenous compound

*sulfate: EC, endogenous compound

*thyroxine: EC, endogenous compound

CAS REGISTRY NO.: (iodothyronine) 29354-16-3; (liothyronine) 6138-47-2,
6893-02-3; (sulfate) 14808-79-8; (thyroxine) 7488-70-2

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ACCESSION NUMBER: 1993:430440 BIOSIS Full-text

DOCUMENT NUMBER: PREV199396085065

TITLE: Gli, a zinc finger transcription factor and oncogene, is
expressed during normal mouse development.

AUTHOR(S): Walterhouse, David; Ahmed, Maqbool; Slusarski, Diane;
Kalamaras, Julie; Boucher, Diane; Holmgren, Robert;
Iannaccone, Philip [Reprint author]

CORPORATE SOURCE: Markey Program Dev. Biol., T239, Northwest. Univ., 303 E.
Chicago Ave., Chicago, IL 60611, USA

SOURCE: Developmental Dynamics, (1993) Vol. 196, No. 2, pp. 91-102.
CODEN: DEDYEI. ISSN: 1058-8388.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Sep 1993

Last Updated on STN: 22 Sep 1993

ABSTRACT: The oncogene GLI is amplified and expressed in some cases of human malignant glioma and undifferentiated childhood sarcoma and is the prototype for a gene family characterized by a highly conserved set of five tandem zinc fingers and a consensus cysteine-histidine link. This zinc finger motif has been shown to bind DNA with sequence specificity and may mediate transcriptional regulation. Since GLI is expressed in embryonal carcinoma cell lines but not in most normal adult tissues and shows significant sequence similarity within its zinc finger domain to cubitus interruptus dominant (ci-D), a Drosophila segmentation gene known to be important in the morphogenesis of the posterior portion of each larval segment, we established the temporal and tissue expression patterns of the mouse homologue of human GLI in day 10 through 18 mouse embryos with Northern blotting, reverse transcriptase coupled PCR, and in situ hybridization. gli transcripts were demonstrated on days 10 through 18 of mouse embryonic development as well as in normal adult uterus, brain, testis, and limb. Tissue expression of gli during gestation was demonstrated in Meckel's precartilaginous mesenchyme, the basis occipitus, rib mesenchymal condensations, primordial vertebral bodies, digital mesenchymal condensations in forefoot and hindfoot plates, the ependymal layer of the spinal cord, and the mesoderm of the gastrointestinal tract. Expression persisted throughout gestation in developing bone and cartilage of the extremities, the ribs, and the vertebral bodies, as well as the gastrointestinal tract mesoderm. These findings support a role for gli family genes in normal craniofacial and digital development in mammals first suggested by the demonstration of translocation breakpoints within the GLI3 gene in families with the Greig cephalopolysyndactyly syndrome and subsequently by reduced gli3 expression in the mouse mutant extra toes. It is surprising that a single gene would be expressed in such a wide range of mesenchymal structures.

CONCEPT CODE: Genetics - Animal 03506
Replication, transcription, translation 10300
Chordate body regions - Head 11304
Chordate body regions - Facial 11306
Chordate body regions - Extremities 11318

Digestive system - Physiology and biochemistry 14004
Bones, joints, fasciae, connective and adipose tissue -
Physiology and biochemistry 18004
Neoplasms - Carcinogens and carcinogenesis 24007
Development and Embryology - General and descriptive
25502

INDEX TERMS: Major Concepts
Development; Genetics; Molecular Genetics (Biochemistry
and Molecular Biophysics); Skeletal System (Movement and
Support); Tumor Biology

INDEX TERMS: Miscellaneous Descriptors
AMNIOTIC FLUID; MECONIUM; SERUM; THYROID HORMONE
METABOLISM; *THYROIDECTOMY*; THYROXINE SULFATE;
3 3' 5-*TRIIODOTHYRONINE SULFATE*

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Muridae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Rodents, Vertebrates

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ACCESSION NUMBER: 1993:159008 BIOSIS Full-text

DOCUMENT NUMBER: PREV199344077808

TITLE: Evidence for thyromimetic effects of 3,5,3'-
triiodothyronine sulfate (T3S) in
hypothyroid rats.

AUTHOR(S): Santini, F.; Hurd, R. E.; Lee, B.; Chopra, I. J.

CORPORATE SOURCE: UCLA Sch. Med., Los Angeles, CA, USA

SOURCE: Clinical Research, (1993) Vol. 41, No. 1, pp. 84A.
Meeting Info.: Joint Meeting of the Western Society for
Clinical Investigation, Western Section American Federation
for Clinical Research, Western Society for Pediatric
Research, Western Region Society for Investigative
Dermatology, and the Western Student Medical Research
Committee. Carmel, California, USA. February 17-20, 1993.
CODEN: CLREAS. ISSN: 0009-9279.

DOCUMENT TYPE: Conference; (Meeting)

LANGUAGE: English

ENTRY DATE: Entered STN: 19 Mar 1993

Last Updated on STN: 19 Mar 1993

CONCEPT CODE: General biology - Symposia, transactions and proceedings
00520

Biochemistry studies - General 10060

Metabolism - Metabolic disorders 13020

Endocrine - Thyroid 17018

INDEX TERMS: Major Concepts
Endocrine System (Chemical Coordination and
Homeostasis); Metabolism

INDEX TERMS: Miscellaneous Descriptors
ABSTRACT

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name

Muridae

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Rodents, Vertebrates